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TRANSACTIONS
OF THE
PATHOLOGICAL SOCIETY
OF PHILADELPHIA.

VOLUME ELEVENTH.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM
SEPTEMBER, 1881, TO JULY, 1883.

EDITED BY

CHAS. B. NANCREDE, M.D.,

PROFESSOR OF GENERAL AND ORTHOPÆDIC SURGERY IN THE PHILADELPHIA POLYCLINIC;
SURGEON TO THE PROTESTANT EPISCOPAL HOSPITAL AND TO ST. CHRISTOPHER'S
HOSPITAL FOR CHILDREN; RECORDER OF THE SOCIETY.

PHILADELPHIA:
PRINTED FOR THE SOCIETY BY T. K. COLLINS.
1884.

THE present volume includes the Transactions of the Society from September, 1881, to June, 1883, inclusive.

The papers of Dr. Sellar, entitled *Some Remarks on the Pathology of Intra-Nasal Hypertrophies* (p. 110); of Dr. J. Solis Cohen, on *Tuberculosis as Manifested in the Larynx* (p. 127); of Dr. C. K. Mills, on *Methodical Examination of the Brain* (p. 231); and Dr. Roberts Bartholow, on *The Trophic System in Pathological Processes* (p. 283), were read by the respective authors at the semi-annual conversational meetings of the Society, they having been appointed to open the proceedings on those evenings.

ERRATA.

In the description of Fig. 2, on page 115, *read* "a posterior hypertrophy in *one* posterior naris," *instead* of "a posterior hypertrophy in *both* posterior naris."

On page 129, middle of 5th line from the top of the page, *read* "Fränkel" *for* "Fränkel."

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Former Presidents.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab.,
elected 1857.

RENÉ LA ROCHE, M.D., elected 1858.

ALFRED STILLÉ, M.D., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M.D., elected 1860 and '63.

J. M. DA COSTA, M.D., elected 1864, '65, and '66.

JOHN H. PACKARD, M.D., elected 1867 and '68.

S. WEIR MITCHELL, M.D., elected 1869.

JOHN ASHHURST, JR., M.D., elected 1870.

JAMES H. HUTCHINSON, M.D., elected 1871 and '72.

WILLIAM PEPPER, M.D., elected 1873.

H. LENOX HODGE, M.D., elected 1876.

S. W. GROSS, M.D., elected 1879.

JAMES TYSON, M.D., elected 1882 and '83.

OFFICERS AND COMMITTEES
OF THE
Pathological Society of Philadelphia.

President.

JAMES TYSON, M.D.
ELECTED OCTOBER, 1883.

ELECTED AT THE ANNUAL MEETING, OCTOBER, 1883.

Vice-Presidents.

E. O. SHAKESPEARE, M.D.,
J. H. C. SIMES, M.D.,
CARL SEILER, M.D.,
J. M. BARTON, M.D.

Secretary.

P. G. SKILLERN, M.D.

Recorder.

C. B. NANCREDE, M.D.

Treasurer.

M. S. FRENCH, M.D.

Curator.

H. F. FORMAD, M.D.

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J. T. ESKRIDGE, M.D.,	C. B. NANCREDE, M.D. (ex-officio).

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H. F. FORMAD, M.D.,	G. DE SCHWEINITZ, M.D.,
J. H. MUSSER, M.D.	

Business Committee.

J. H. NEFF, M.D.,	J. M. BARTON, M.D.,
J. H. MUSSER, M.D.,	W. E. HUGHES, M.D.

LIST OF MEMBERS.

Members marked O. M. are of those originating the Society.

Members marked N. R. are Non-resident.

Elected

- 1857 AGNEW, D. HAYES, Rhea Barton Professor of Surgery in the University of Pennsylvania, 1611 Chestnut Street. (O. M.)
- 1871 ALISON, ROBERT H., Physician to the Hospital for Nervous Diseases, 250 South Seventeenth Street.
- 1864 ALLEN, HARRISON, resigned December 11, 1879.
- 1869 ALLIS, OSCAR H., resigned February 24, 1881.
- 1865 ANDREWS, THOMAS H., 1117 Spruce Street.
- 1871 ASHBRIDGE, WILLIAM, resigned December 11, 1879.
- 1867 ASHHURST, FRANCIS, Mount Holly, New Jersey. (N. R.)
- 1861 ASHHURST, JOHN, JR., Professor of Clinical Surgery in the University of Pennsylvania, 2000 West De Lancey Place.
- 1863 ASHHURST, SAMUEL, Surgeon to the Children's Hospital, 2308 West De Lancey Place.
- 1871 ATLEE, WASHINGTON, L., Jr., forfeited membership by non-payment of dues, December 10, 1874.
- 1866 BACHE, T. HEWSON, resigned December 27, 1883.
- 1878 BAER, B. F., resigned.
- 1876 BARR, J. W., forfeited membership by non-payment of dues, December 8, 1881.
- 1880 BARTHOLOW, ROBERTS, Professor of Materia Medica in the Jefferson Medical College, 1509 Walnut Street.
- 1867 BARTLES, WM. H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)
- 1883 BARTON, ISAAC, 128 South Fifteenth Street.
- 1869 BARTON, J. M., Surgeon to the German and Jefferson College Hospitals, 1344 Spruce Street.
- 1873 BEECHER, A. C. W., 523 South Ninth Street.
- 1870 BELL, J. R. F., forfeited membership by non-payment of dues, December 10, 1874.
- 1871 BENNETT, WM. H., 332 South Fifteenth Street.
- 1863 BENTON, C. H. (N. R.)
- 1875 BERNARDY, EUGENE P., resigned September 22, 1881.
- 1870 BERTHOLET, ROBERT MORRIS, died 1882.
- 1871 BETTS, THOMAS, Branchtown, Pa. (N. R.)
- 1860 BISHOP, C. S., died.
- 1866 BLACK, J. J., New Castle, Delaware. (N. R.)

Elected

- 1863 BOARDMAN, C. H., St. Paul, Minnesota. (N. R.)
- 1880 BØNNING, HENRY C., forfeited membership by non-payment of dues, April 14, 1882.
- 1857 BOKER, CHARLES S., Surgeon to St. Joseph's Hospital, 1622 Chestnut Street. (O. M.)
- 1868 BOLLES, LUCIUS S., died August 15, 1873.
- 1883 BOYD, GEO. M., 238 West Logan Square.
- 1877 BRADFORD, T. HEWSON, resigned December 23, 1880.
- 1873 BRAY, DANIEL, forfeited membership by non-payment of dues, December 8, 1881.
- 1883 BRENNIG, P. B., Bethlehem, Pennsylvania. (N. R.)
- 1857 BRINTON, JOHN H., Professor of the Practice of Surgery and Clinical Surgery in the Jefferson Medical College, 1423 Spruce Street. (O. M.)
- 1874 BRUBAKER, A. P., 1210 Race Street.
- 1874 BRUEN, E. T., Physician to the Philadelphia Hospital, 1531 Chestnut Street.
- 1871 BUCK, WM. PENN, resigned October 23, 1873.
- 1867 BURNETT, CHAS. H., resigned December 8, 1881.
- 1872 CATHCART, JAMES H., died March 5, 1874.
- 1879 CATHCART, T. H., died March 28, 1882.
- 1871 CHESTON, C. MORRIS, West River, Maryland. (N. R.)
- 1865 CHESTON, D. MURRAY, resigned December 13, 1883.
- 1871 CLARK, LEONARDO S., resigned November 29, 1883.
- 1866 CLEEMANN, RICHARD A., Physician to St. Mary's Hospital, 2135 Spruce Street.
- 1872 COHEN, J. SOLIS, Professor of Diseases of the Throat and Chest in the Philadelphia Polyclinic and School for Graduates, 1431 Walnut Street.
- 1883 COHEN, S. SOLIS, 1431 Walnut Street.
- 1869 CORBIT, WILLIAM B., Washington, D. C. (N. R.)
- 1877 CRUCE, R. B., 731 North Seventeenth Street.
- 1871 CURTIN, ROLAND G., Physician to the Philadelphia Hospital, 22 South Eighteenth Street.
- 1857 DA COSTA, J. M., Professor of the Theory and Practice of Medicine in the Jefferson Medical College, 1700 Walnut Street. (O. M.)
- 1859 DARBY, J. T., New York City. (N. R.)
- 1876 DARLINGTON, W. L. (N. R.)
- 1857 DARRACH, JAMES, 5094 Green Street, Germantown. (O. M.)
- 1868 DARRACH, WILLIAM, died January 28, 1881.
- 1882 DAVIS, G. D., 1817 Mount Vernon Street.
- 1877 DEAVER, R. W., 5075 Main Street, above High, Germantown.

Elected

- 1882 DERGUM, F. X., 636 North Eighth Street.
 1882 DE SCHWEINITZ, G. E., 1330 Spruce Street.
 1883 DUNN, W. G., West Chester. (N. R.)
 1866 DUER, EDWARD L., Professor of Diseases of Women and Children in the Philadelphia Polyclinic and School for Graduates, 1704 Arch Street.
 1870 DUHRING, L. A., resigned November 9, 1882.
 1876 DULLES, C. W., 3932 Locust Street.
 1860 DUNTON, W. R., 5059 Germantown Avenue, Germantown.
 1883 DUNMIRE, G. B., 1116 Arch Street.

 1883 EDWARDS, J. E., 115 South Seventh Street.
 1882 EDWARDS, W. A., 1210 Spruce Street.
 1875 ENGEL, HUGO, forfeited membership by non-payment of dues, February 23, 1882.
 1876 ESKRIDGE, J. T., Physician to the Jefferson College Hospital, 1614 North Sixteenth Street.

 1881 FENTON, T. H., 1335 Arch Street.
 1857 FISCHER, EMIL, southeast corner Sixth and Brown Streets. (O. M.)
 1863 FISH, AUGUSTINE H., died August 3, 1872.
 1876 FISHER, HENRY M., Physician to the Episcopal Hospital, 919 Walnut Street.
 1857 FORBES, WILLIAM S., one of the Surgeons to the Episcopal Hospital, 1405 Locust Street. (O. M.)
 1869 FORD, WILLIAM H., 1622 Summer Street.
 1878 FORMAD, H. F., 105 South Thirty-fourth Street.
 1871 FOX, CHARLES, resigned December 10, 1874.
 1880 FRENCH, M. S., 1423 Walnut Street.

 1883 GARRET, E. F., 5043 Germantown Avenue, Germantown.
 1868 GARRETSON, JAMES E., resigned November 22, 1883.
 1871 GERHARD, GEORGE S., Ardmore. (N. R.)
 1870 GETCHELL, FRANK H., 1432 Spruce Street.
 1877 GILLIARD, LOUIS, died November, 1878.
 1882 GODEY, H. E., N. E. corner Nineteenth and Spruce Streets.
 1877 GOLDSBOROUGH, C. B. (N. R.)
 1866 GOODELL, WILLIAM, Clinical Professor of Diseases of Women in the University of Pennsylvania, Preston Retreat, Twentieth and Hamilton Streets.
 1868 GOODMAN, H. ERNEST, resigned February 23, 1882.
 1868 GRAHAM, JAMES, resigned January 9, 1879.
 1867 GRIER, MATTHEW J., 1531 Spruce Street.
 1883 GRIFFITH, J. P. C., 110 South Eighteenth Street.

Elected

- 1868 GROSS, FERDINAND H., one of the Surgeons to the German Hospital, 1416 Girard Avenue.
- 1857 GROSS, SAMUEL D., Emeritus Professor of Surgery in the Jefferson Medical College, southeast corner Eleventh and Walnut Streets. (O. M.) Died May 6, 1884.
- 1858 GROSS, SAMUEL W., Professor of the Principles of Surgery and Clinical Surgery in the Jefferson Medical College, 1112 Walnut Street.
- 1869 GROVE, JOHN H., one of the Surgeons to St. Mary's Hospital, 1330 Arch Street.
- 1872 GRUEL, THEODORE H. E., 612 Spruce Street.
- 1874 GUITÉRAS, JOHN. (N. R.)
- 1871 HALE, GEORGE J., forfeited membership by non-payment of dues, March 23, 1876.
- 1857 HALL, A. DOUGLASS, resigned October 9, 1873. (O. M.)
- 1875 HAND, FRANK C., died September 1, 1881.
- 1866 HARE, HORACE BINNEY, died March 21, 1879.
- 1868 HARGADINE, R. W. (N. R.)
- 1859 HARLAN, GEORGE C., resigned January 12, 1882.
- 1874 HARRIS, CHARLES M., 330 South Sixteenth Street.
- 1858 HARRIS, ROBERT P., resigned January 9, 1873.
- 1881 HART, R. M., forfeited membership by non-payment of dues.
- 1857 HARTSHORNE, EDWARD, 331 South Broad Street. (O. M.)
- 1859 HARTSHORNE, HENRY, resigned October 12, 1864.
- 1867 HASSLER, FERDINAND A., resigned February 9, 1871.
- 1869 HATFIELD, NATHAN L., one of the Visiting Surgeons to the Philadelphia Hospital, 501 Franklin Street.
- 1868 HAYS, I. MINIS, resigned January 13, 1881.
- 1879 HAZLEHURST, S. F. (N. R.)
- 1873 HEARN, JOSEPH, one of the Surgeons to the Jefferson College and Philadelphia Hospitals, 324 Catharine Street.
- 1870 HENRY, FREDERICK P., Professor of Pathology and Microscopy in the Philadelphia Polyclinic and School for Graduates, one of the Physicians to the Episcopal Hospital, 721 Pine Street.
- 1866 HERBERT, THEODORE. (N. R.)
- 1879 HESS, ROBERT, resigned January 12, 1882.
- 1857 HEWSON, ADDINELL (O. M.), forfeited membership by non-payment of dues, December 22, 1881.
- 1880 HEWSON, A., JR., 1304 Pine Street.
- 1874 HICKMAN, N. B., forfeited membership by non-payment of dues, December 8, 1881.
- 1883 HINSDALE, GUY, 202 South Forty-first Street.
- 1858 HODGE, H. LENOX, died June 10, 1881.
- 1883 HOELLY, G. H., Camden, N. J.

Elected

- 1871 HOFFMAN, WASHINGTON ATLEE, died September 20, 1874.
- 1860 HOPKINS, HENRY ST. GEORGE. (N. R.)
- 1876 HOPKINS, W. B., resigned October 14, 1880.
- 1877 HORWITZ, THEODORE, died December 12, 1877.
- 1870 HOUSTON, JAMES P. S., Savannah, Georgia. (N. R.)
- 1866 HOWE, HERBERT M., resigned February 28, 1878.
- 1859 HÓYT, WILLIAM D. (N. R.)
- 1880 HUGHES, D. E., 52 South Thirteenth Street.
- 1882 HUGHES, W. E., 3724 Baring Street.
- 1879 HUIDEKOPER, R. S., resigned October 28, 1880.
- 1857 HUMPHRIES, GEORGE H., New York City. (O. M., N. R.)
- 1868 HUNTER, CHARLES T., resigned October 14, 1880. Died 1884.
- 1870 HUTCHINS, E. R. (N. R.)
- 1858 HUTCHINSON, JAMES H., one of the Physicians to the Pennsylvania Hospital, 2019 Walnut Street.

- 1866 INGHAM, JAMES V., resigned December 11, 1879.
- 1876 INGRAM, T. D., forfeited membership by non-payment of dues, December 8, 1881.

- 1874 JAMISON, EDWARD, St. Louis, Mo. (N. R.)
- 1866 JENKS, WILLIAM F., died October 31, 1881.
- 1875 JOHNSON, RUSSELL H., resigned December 8, 1881.
- 1883 JUDD, L. D., 3603 Powelton Avenue.

- 1859 KANE, JOHN K., Wilmington, Delaware. (N. R.)
- 1874 KEATING, JOHN M., resigned December 22, 1881.
- 1857 KEATING, WILLIAM V. (O. M.), forfeited membership by non-payment of dues, September 14, 1864.
- 1857 KEALER, WILLIAM, resigned January 28, 1875. (O. M.)
- 1866 KEEN, WILLIAM W., Professor of Surgery, Woman's Medical College, 1729 Chestnut Street.
- 1875 KELLY, JOSEPH V., 4257 Main Street, Manayunk.
- 1884 KILDUFFE, ROBERT, 754 South Twelfth Street.
- 1858 KING, WILLIAM, died.
- 1879 KIRKBRIDE, J. J., 35 South Nineteenth Street.
- 1869 KNIGHT, SAMUEL R., resigned October 28, 1880.

- 1867 LAMBDIN, ALFRED C., forfeited membership by non-payment of dues, December 10, 1874.
- 1871 LANDIS, HENRY G., Niles, Ohio. (N. R.)
- 1872 LA ROCHE, C. PERCY, Nice, France. (N. R.)
- 1857 LA ROCHE, RENÉ. (O. M.) Died December 9, 1872.
- 1879 LEACH, ALONZO L., forfeited membership by non-payment of dues, December 22, 1881.

Elected

- 1869 LEAMAN, HENRY, 1031 Vine Street.
 1860 LEE, CHARLES C., New York City. (N. R.)
 1882 LEE, J. G., resigned September, 1883.
 1861 LEEDOM, JOHN M., Pulaski Avenue, Germantown.
 1857 LEVICK, JAMES J., resigned October 13, 1861. (O. M.)
 1868 LEWIS, FRANCIS W., 2016 Spruce Street.
 1869 LEWIS, FRED. W., died 1873.
 1875 LEWIS, MORRIS J., one of the Physicians to the Episcopal Hospital,
 1216 Walnut Street.
 1857 LEWIS, SAMUEL, Aldine Hotel. (O. M.)
 1877 LITTLE, WILLIAM S., 215 South Seventeenth Street.
 1859 LIVEZY, EDWARD, forfeited membership by non-payment of dues,
 September 14, 1864.
 1876 LODER, P. E., forfeited membership by non-payment of dues, Febru-
 ary 9, 1882.
 1873 LONGENECKER, JEROME, forfeited membership by non-payment of
 dues, 1875.
 1875 LONGSTRETH, MORRIS, one of the Physicians to the Pennsylvania
 Hospital, 1416 Spruce Street.
 1870 LOUGHLIN, J. ENEU, forfeited membership by non-payment of dues,
 May 9, 1878.

 1882 MACCONNELL, W. G., 250 South Tenth Street.
 1874 MANN, CHARLES H. (N. R.)
 1867 MARKOE, JAMES, died March, 1884.
 1868 MARTIN, GEORGE. (N. R.)
 1874 MATTESON, CHARLES C., died.
 1865 MAURY, FRANK F., died 1879.
 1870 MAXWELL, J. GORDON, forfeited membership by non-payment of dues,
 December 10, 1874.
 1866 MCARTHUR, JOHN A., forfeited membership by non-payment of dues,
 December 8, 1881.
 1861 MCCALL, C. A., resigned.
 1880 MCCLELLAN, G., 1352 Spruce Street.
 1868 MCCLURE, W. WALLACE, resigned September 10, 1878.
 1873 MCCOY, A. W., 1415 Walnut Street.
 1884 MCILVAINE, E. S., 4025 Chestnut Street.
 1864 MEARS, J. EWING, one of the Surgeons to St. Mary's Hospital, 1429
 Walnut Street.
 1873 MEIGS, ARTHUR V., one of the Physicians to the Pennsylvania Hos-
 pital, 1322 Walnut Street.
 1857 MEIGS, JOHN FORSYTH, died December 16, 1882. (O. M.)
 1874 MILLER, C. K. I., died 1878.
 1882 MILLER, D. J. M., 130 South Fifteenth Street.

Elected

- 1878 MILLS, C. K., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Neurologist to the Philadelphia Hospital; Lecturer on Mental Diseases in the University of Pennsylvania, 113 South Nineteenth Street.
- 1884 MITCHELL, J. K., 1524 Walnut Street.
- 1857 MITCHELL, S. WEIR, one of the Physicians to the Hospital for Nervous Diseases, 1524 Walnut Street. (O. M.)
- 1876 MONTGOMERY, E. C., resigned January 11, 1883.
- 1857 MOREHOUSE, GEORGE R., 227 South Ninth Street.
- 1884 MORRISON, WM. H., Holmesburg, Pa. (N. R.)
- 1857 MORTON, THOMAS GEORGE (O. M.), forfeited membership by non-payment of dues, September 14, 1864.
- 1859 MOSS, WILLIAM, Chestnut Avenue, Chestnut Hill.
- 1871 MUHLENBERG, FRANK, 1911 Chestnut Street.
- 1869 MULLER, AUGUST F., 4544 Germantown Avenue.
- 1880 MUSSER, JOHN H., Chief of Medical Dispensary, University Hospital, 3705 Powelton Avenue.
- 1873 MUSSER, MILTON B., Fortieth and Locust Streets.
- 1869 MUSTIN, J. BURTON, died 1871.
- 1869 NANCREDE, CHARLES B., Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic and School for Graduates, Surgeon to the Episcopal Hospital, Surgeon to St. Christopher's Hospital for Children, 2109 Pine Street.
- 1879 NEFF, J. S., 214 South Fifteenth Street.
- 1880 NEWCOMET, H. W., 1327 Girard Avenue.
- 1872 NEWSHAM, STANLEY P., resigned December 23, 1881.
- 1868 NORRIS, HERBERT, resigned January 8, 1880.
- 1868 NORRIS, ISAAC, resigned November 12, 1874.
- 1869 NORRIS, J. C., resigned February 10, 1876.
- 1861 NORRIS, WILLIAM F., Clinical Professor of Ophthalmology in the University of Pennsylvania, 1526 Locust Street.
- 1875 O'HARA, MICHAEL, 31 South Sixteenth Street.
- 1874 OSGOOD, HAMILTON. (N. R.)
- 1857 PACKARD, JOHN H., Surgeon to the Pennsylvania and St. Joseph's Hospitals, 1928 Spruce Street. (O. M.)
- 1868 PANCOAST, WILLIAM H., Professor of Anatomy in the Jefferson Medical College, 1100 Walnut Street.
- 1873 PARISH, WILLIAM H., Professor of Anatomy in the Woman's Medical College, 324 South Seventeenth Street.
- 1875 PARKS, EDWARD L. (N. R.)
- 1867 PARRY, JOHN S., died 1876.
- 1870 PAUL, COMEGYS, Belvidere, N. J. (N. R.)
- 1875 PAUL, JAMES, resigned January 22, 1880.

Elected

- 1857 PENROSE, R. A. F. (O. M.), forfeited membership by non-payment of dues, September 14, 1864.
- 1865 PEPPER, GEORGE, died September 14, 1872.
- 1865 PEPPER, WILLIAM, Professor of the Theory and Practice of Medicine in the University of Pennsylvania, 1811 Spruce Street.
- 1879 PERKINS, F. M., 1428 Pine Street.
- 1883 PIERSOL, G. A., 1110 Spring Garden Street.
- 1870 PORTER, WILLIAM G., JR., one of the Surgeons to the Presbyterian Hospital, 1223 Spruce Street.
- 1878 RAUB, J. A., 927 North Tenth Street.
- 1888 RAUGHTER, C. A., Harrisburg, Pa. (N. R.)
- 1888 REED, B., Atlantic City, N. J. (N. R.)
- 1874 REED, HENRY B., resigned December 9, 1880.
- 1859 REED, THOMAS B., one of the Surgeons to the Presbyterian Hospital, 1427 Walnut Street.
- 1871 REX, GEORGE A., 2118 Pine Street.
- 1869 REX, OLIVER P., forfeited membership by non-payment of dues, December 10, 1874.
- 1877 REYNOLDS, A. S., forfeited membership by non-payment of dues, February 9, 1882.
- 1864 RHODES, EDWARD, died January 16, 1871.
- 1878 RICH, T. C., resigned January 13, 1881.
- 1869 RICHARDSON, ELLIOTT, resigned December 8, 1881.
- 1863 RICHARDSON, JOSEPH G., one of the Visiting Physicians to the Presbyterian Hospital, 3238 Chestnut Street.
- 1857 RICHARDSON, T. G., New Orleans, La. (O. M., N. R.)
- 1882 RIDGE, J. M., Camden, N. J.
- 1876 RISLEY, S. D., Lecturer on Ophthalmoscopy in the University of Pennsylvania, 1630 Walnut Street.
- 1868 RITZ, CHARLES M. (N. R.)
- 1880 ROBERTS, A. S., Instructor in Orthopædics in the University of Pennsylvania, Surgeon to the Philadelphia Hospital, 131 South Fifteenth Street.
- 1876 ROBERTS, J. B., Professor of General and Operative Surgery in the Philadelphia Polyclinic and School for Graduates, Surgeon to St. Mary's Hospital, 1118 Arch Street.
- 1876 ROLAND, O. (N. R.)
- 1874 RONALDSON, WILLIAM D., 4017 Locust Street.
- 1877 RUDOLPH, B. J., forfeited membership by non-payment of dues, February 23, 1882.
- 1867 SANTEE, EUGENE J., 532 North Sixth Street.
- 1863 SAVERY, WILLIAM, resigned February 11, 1869.
- 1869 SCHELL, H. S., resigned December 8, 1881.
- 1874 SEILER, CARL, 1346 Spruce Street.

Elected

- 1870 SEYFERT, THEODORE H., resigned December 27, 1877.
- 1870 SHAFFNER, CHARLES, 251 South Thirteenth Street.
- 1876 SHAKESPEARE, E. O., Ophthalmic Surgeon to the Philadelphia Hospital, 1336 Spruce Street.
- 1869 SHAPLEIGH, ELISHA B., 658 North Eighth Street.
- 1868 SHERWOOD, THOMAS H., forfeited membership by non-payment of dues, December 10, 1874.
- 1878 SHOEMAKER, J. V., 1031 Walnut Street.
- 1871 SIMES, J. H. C., Professor of Genito-Urinary Diseases in the Philadelphia Polyclinic and School for Graduates, Surgeon to the Episcopal Hospital, 2033 Chestnut Street.
- 1868 SINKLER, WHARTON, one of the Physicians to the Hospital for Nervous Diseases, 1534 Pine Street.
- 1881 SKILLERN, P. G., 429 South Broad Street.
- 1881 SKILLERN, S. R., 3416 Baring Street.
- 1883 SIMON, J. W., 340 South Fifteenth Street.
- 1858 SMITH, A. H., Lecturer on Obstetrics at the Philadelphia Lying-in Charity, 1419 Walnut Street.
- 1863 SMITH, E. A., Aldine Hotel.
- 1859 SMITH, F. G., resigned November 13, 1873. Died April 6, 1878.
- 1876 SMITH, R. M., resigned December 9, 1880.
- 1875 SMITH, STANLEY. (N. R.)
- 1876 SMYTHE, F. G., died 1879.
- 1871 SPARKS, GEORGE W., forfeited membership by non-payment of dues, December 10, 1874.
- 1864 SPOONER, EDWARD A., forfeited membership by non-payment of dues, December 10, 1874.
- 1871 STARR, LOUIS, resigned October 14, 1880.
- 1883 STILES, G. M., Conshohocken, Pa.
- 1857 STILLÉ, ALFRED, Professor Emeritus of Theory and Practice of Medicine in the University of Pennsylvania, 3900 Spruce Street. (O. M.)
- 1870 STRAWBRIDGE, GEORGE, resigned June 10, 1875.
- 1882 STRITTMATTER, J. P., 1232 North Fifth Street.
- 1869 STRYKER, SAMUEL S., one of the Obstetricians to the Philadelphia Hospital, 3713 Walnut Street.
- 1879 TAYLOR, J. M., northwest corner Twenty-second Street and St. Alban's Place.
- 1866 THOMAS, CHARLES H., 1807 Chestnut Street.
- 1868 THOMSON, WILLIAM, one of the Surgeons to Wills Ophthalmic Hospital, 1502 Locust Street.
- 1870 TINKHAM, J. H., United States Navy. (N. R.)
- 1866 TOWNSEND, GEORGE D. (N. R.)
- 1868 TOWNSEND, R. M., died.

Elected

- 1864 TURNER, A. P. (N. R.)
- 1863 TUTT, CHARLES PENDLETON, died May 11, 1866.
- 1863 TYSON, JAMES, Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, 1506 Spruce Street.
- 1883 ULRICH, W. B., Chester, Pa. (N. R.)
- 1871 VALDIVIESO, R. (N. R.)
- 1870 VAN HARLINGEN, ARTHUR, resigned December 8, 1881.
- 1879 VANVALZAH, W. W., resigned October 26, 1882.
- 1874 WARDER, WM. H., one of the Obstetricians to the Philadelphia Hospital, 810 North Broad Street.
- 1867 WATSON, E. W., 201 North Twentieth Street.
- 1866 WEIGHTMAN, JOHN F., resigned April 27, 1871.
- 1876 WEIR, A. H., resigned April 14, 1881.
- 1875 WEST, FRANKLIN, died.
- 1882 WETHERILL, H. M., Pennsylvania Hospital for the Insane.
- 1875 WHELEN, ALFRED, resigned December 23, 1880.
- 1873 WHITE, J. WILLIAM, one of the Visiting Surgeons to the Philadelphia Hospital, 222 South Sixteenth Street.
- 1868 WILLARD, DE FOREST, Lecturer on Orthopædic Surgery in the University of Pennsylvania, 1818 Chestnut Street.
- 1865 WILLIAMS, HORACE, resigned December 11, 1879.
- 1875 WILLIAMSON, JESSE, resigned October 13, 1881.
- 1867 WILSON, ELLWOOD, resigned.
- 1882 WILSON, C. M., 1517 Walnut Street.
- 1880 WILSON, H. A., resigned December 30, 1882.
- 1869 WILSON, JAMES C., one of the Visiting Physicians to the Philadelphia and Jefferson College Hospitals, 1437 Walnut Street.
- 1867 WILSON, JAMES F., 1010 Race Street.
- 1869 WILSON, J. H. (N. R.)
- 1873 WINSLOW, W. H., resigned December 28, 1876.
- 1864 WISTAR, THOMAS, resigned February 13, 1867.
- 1875 WOLFORD, W. S., 133 South Thirteenth Street.
- 1863 WOOD, HORATIO C., Jr., forfeited membership by non-payment of dues, March 23, 1876.
- 1883 WOODBURY, F., 218 South Sixteenth Street.
- 1865 WOODS, D. FLAVEL, one of the Physicians to the Presbyterian Hospital, 1501 Spruce Street.
- 1857 WOODWARD, J. J., U. S. A. (O. M., N. R.), died 1884.
- 1872 WORTHINGTON, DAVID J., forfeited membership by non-payment of dues, February 9, 1882.
- 1876 ZIEGLER, G. W. (N. R.)

CORRESPONDING MEMBERS.

- 1882 **ARNOLD, J. W. S.**, New York, Professor Emeritus of Physiology and Histology in the University of the City of New York.
- 1866 **BUMSTEAD, FREEMAN J.** Died 1879.
- 1859 **CLARK, ALONZO**, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York. Died 1884.
- 1882 **CONNER, P. S.**, Professor of Anatomy and Clinical Surgery in the Medical College of Ohio, Cincinnati, Ohio.
- 1859 **DALTON, J. C.**, Professor of Physiology in the College of Physicians and Surgeons, New York.
- 1882 **DELAFIELD, FRANCIS**, Adjunct Professor of Pathology and Practical Medicine in the College of Physicians and Surgeons, New York.
- 1860 **ELLIS, CALVIN**, Professor of Theory and Practice of Medicine in the Medical Department of Harvard University, Boston, Mass.
- 1882 **FITZ, REGINALD H.**, Shattuck Professor of Pathological Anatomy in the Medical Department of Harvard University, Boston, Mass.
- 1858 **FLINT, AUSTIN, SR.**, Professor of Theory and Practice of Medicine in Bellevue Hospital Medical College, New York.
- 1858 **HAMMOND, WILLIAM A.**, Member of the National Academy of Sciences, New York.
- 1860 **ISAACS, CHARLES E.**, Brooklyn. Died June 16, 1860.
- 1878 **JACKSON, J. B. S.**, Boston. Died 1879.
- 1882 **JANEWAY, EDWARD G.**, Professor of Pathological Anatomy and Clinical Medicine, and Associate Professor of the Principles and Practice of Medicine in the Bellevue Hospital Medical College, New York.
- 1860 **REEVES, JAMES E.**, Wheeling, West Virginia.
- 1861 **ROKITANSKY, CARL.** Died 1878.
- 1882 **SEGUIN, E. C.**, New York.
- 1882 **WARREN, J. COLLINS**, Assistant Professor of Surgery in the Medical Department of Harvard University, Boston, Mass.
- 1860 **WATSON, JOHN**, New York. Died June 3, 1869, æt. 56.
- 1882 **WHITTAKER, JAMES T.**, Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medical College of Ohio, Cincinnati, Ohio.
- 1859 **WORTHINGTON, WILMER**, West Chester, Pennsylvania. Died 1873.

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REPORT.

I. THE OSSEOUS SYSTEM.

1. *Osteoid chondroma of the pelvis.*

Exhibited by Dr. H. F. FORMAD.

THE specimen exhibited is a portion from an enormous tumor removed post-mortem by Dr. J. T. Ullom, of Rogersville, Greene County, Pennsylvania.

The following history was kindly furnished by Miss Jennie Teagarden, medical student :—

Mrs. Moore, of Greene County, Pennsylvania, married, æt. 36, height five feet three inches, weight in health one hundred and twenty-five pounds. Mother of three children ; two are now living. The youngest child, born March, 1878, is healthy and well developed. Medical aid was first called during the summer of 1873, after having noticed a small hard lump in right iliac fossa ; the growth about the size of a hickory-nut ; no treatment ; patient advised to let it alone.

One year later, another physician, Dr. J. T. Ullom, was called. On examination, he found a tumor about the size of a walnut, firmly fixed, causing slight lameness, otherwise giving no trouble. The tumor was thought at the time to be fibro-cartilaginous ; no special treatment. The general health not impaired, but tumor rapidly increasing in size. During the year 1876, a dull, aching pain in the parts ; hip-joint became ankylosed. Patient did not lose flesh ; menstrual function normal until last six months of life, then became scanty and irregular ; three months before death menses disappeared entirely. About the same period, micturition became extremely difficult and painful ; constipation during last year of life. At time of birth of last child, the tumor filled one-half of the pelvis, gave great trouble in delivery, a slow labor exhausting patient very much. During last two years was obliged to lie in bed continually, the bed being a very low couch ; could not rise up or move the affected limb. The appetite continued, digestion seemed

unimpaired, and patient did not suffer greatly until two weeks preceding death. Defecation and urination then became almost impossible; catheter could not be passed. The extremities became swollen and discolored, small blisters appearing upon the one on affected side. A small growth resembling the large one made its appearance upon the ankle of the other extremity.

The mind remained clear. Death, resulting from mechanical obstruction of bowels and bladder, occurred August 7, 1881.

Autopsy, ten hours after death.

The upper portion of the body presented no unusual appearance; lower limbs very much swollen; right limb presented a gangrenous appearance. A tumor covering whole of outer side of right hip, extending from median line to spine, and from knee to umbilicus in front and to ribs behind.

Longest diameter, forty-nine inches. From inside of thigh around nates, fifty-three inches.

Middle third of thigh, forty-six inches. Circumference of abdomen above tumor, thirty-five inches.

From crest of ilium around right natis, twenty-eight inches. Surface presented nodulated and irregular appearance. Dissection presented same appearance; integument not adherent; separated readily; immediately beneath integument numerous cartilaginous growths formed singly and in clusters; some of these creak under the knife, a serous fluid escaping; no resemblance to muscle or bloodvessels. Deep dissection showed the whole joint involved, osseous growths, and cysts from which a jelly-like fluid escaped, in some parts of a dark grumous color; middle and upper part of femur also involved. No muscles, nerves, bloodvessels, or bones; all appropriated to the abnormal growth. In abdominal cavity a large quantity of a gelatinous substance. The uterus normal, also the left ovary; right barely recognizable. Weight of whole tumor seventy-five and one-fourth pounds. No examination made of thoracic cavity. Death resulted from mechanical obstruction of bladder and bowels. Later diagnosis, enchondroma. The very early history not satisfactory; the post-mortem unavoidably made with haste.

Dr. Gross said that he thought, from the description of the tumor given by Dr. Formad, the growth should be classed as an *ossifying osteoid chondroma*.

Dr. Formad said that it was true that bony plates were found; also calcifications; but, as Virchow has pointed out, both ossification and calcification are the invariable and natural fate of the osteoid chon-

dromata. Virchow has described them as benign, although metastases of the growths may occur. It is worthy of note that, with the exception of three or four cases, osteoid chondromata of the pelvis *which occurred in multiparous women* all followed fractures or injuries.

Dr. Gross had not read Virchow's account of these tumors for several years, but thought that Dr. Formad was wrong, as Virchow distinctly states that they are of a suspicious nature, and relates a case in which metastatic growths had formed in the lungs and pleura. He thought all other German authors agreed in speaking of the great bulk attained by these growths and their extreme malignancy.

Dr. Formad thought that metastasis was not always a proof of malignancy. No one considers the osteoid chondroma or the pure chondroma malignant, although numerous cases of the latter tumor are on record in which metastasis is said to have occurred.

Dr. Gross opposed this view, and said that he doubted if a *pure* chondroma of any organ ever resulted in metastasis, instancing the recent analysis of the cases of so-called malignant chondromata of the testicle by Mr. Bultin in support of this view.

Dr. Formad then spoke of chondroma of the testicle where the cartilaginous substance was disposed in the form of little cylinders wedged into the lymph-spaces, which might readily become dislodged and give rise to metastatic growths in the lungs. It has been also experimentally proved by Henry Wile that particles of any normal tissue transplanted to the lungs by means of the jugular vein grow and develop in precisely the same manner as malignant metastatic tumors. He did not deny that in many cases of cartilaginous growths in the lungs the question of their local origin from the bronchial cartilages might not fairly arise. The readiness with which the cartilaginous cylinders might be dislodged was pressed by Virchow.

Dr. Seiler asked whether the apparent difference between the different speakers could not be explained by stating that in enchondroma of any organ similar multiple growths were found which perhaps had no connection as cause and effect.

Dr. Nancrede referred to the fact of the common association of cartilage with sarcomatous, myxomatous, and other growths, especially in testicular growths. Of course microscopical examination of a metastasis of such a mixed tumor would sometimes perhaps reveal cartilage alone, when the mistake as to chondromatous metastases might readily arise unless an exhaustive examination of the primary growth had been made.

January 26th, 1882.

2. *Gunshot wound of the mastoid process of the temporal bone, involving the lateral sinus; insignificant hemorrhage.*

Exhibited by Dr. C. B. NANCREDE.

H. M., æt. *circa* 25 years, was admitted into the male surgical wards of the Episcopal Hospital under my charge on January 1, having accidentally shot himself the evening before with a small 22-calibre ball behind the right ear. Trifling bleeding had occurred, but little if any shock, and he walked unassisted to the hospital. No motor or sensory trouble was ever noted. On examining him carefully, the probe passed upward, inward, and backward through the mastoid process to such a depth as to convince me that the lateral sinus must be injured, especially as rather free venous bleeding ensued upon the withdrawal of the probe. Marked meningeal irritation ensued, with flushed face, congested eyes, high pulse and temperature, which was relieved by appropriate treatment, only to be followed by a pyæmic chill on January 11, when the mastoid process was trephined to give freer exit to the rather profuse discharge. A portion of the ball was found between the integument and the bone, but the main portion of the ball was not found. Some relief ensued, but on the 12th and 15th the chills recurred, and he died January 16, in a markedly typhoid condition. The post-mortem examination showed that the ball lay partly within the sinus, partly between the bone and the upraised sinus-wall. Indeed, on opening the sinus, which was partially filled with ante-mortem and fully by post-mortem clot, the ball rolled out. Whether the ball originally penetrated into the sinus at all, whether it partially entered and the opening became larger by ulceration, or whether the aperture was wholly due to the latter process, could not be determined. One or two small secondary abscesses were detected in the lungs.

Jan. 26th, 1882.

3. *Tumor of the alveolar process of the lower jaw.*

Exhibited by Dr. J. B. ROBERTS.

A young woman, aged about 20 years, had had for some months a flattened and ulcerated growth on the right side of lower alveolus behind the lower teeth. The tumor overlapped the borders of the gum, and had a fungoid appearance. There was no special pain, no glandular

involvement, and the general health was very good. I cut away the upper portion by an elliptical incision with the scalpel, and finding the last molar displaced inward, and evidently in close relation with the tumor, seized it with the forceps in order to extract it. To my surprise, the tooth was easily lifted from the bone, and brought with it the remainder of the growth,—about three-quarters of an inch or an inch in diameter,—and left a cavity in the jaw large enough to insert my finger. The surfaces of the cavity and the rough edges of bone were thoroughly cut away by the burr of the surgical engine. On the tenth day the patient was discharged from my care to return to her home in the centre of the State, although there was still a little stiffness of the jaws from swelling and an incompletely healed wound. I have not heard from her since that time, which is about five years ago.

Report of the Committee on Morbid Growths.—"A microscopical section of the tumor shows, upon examination, the histological structure of fibrous tissue. This tissue is seen in different parts of the section in its various stages of development,—young and embryonal, as indicated by embryonic cells; myxomatous, by stellate and fusiform cells, with a soft, gelatinous, intercellular substance; and mature, by a distinct fibrillar substance separating spindle-shaped cells. The bloodvessels possess evident walls. The growth is a fibroma."

April 12th, 1882.

4. *Specimens from an excision of the hip-joint where death resulted from tubercular meningitis.*

Presented by Dr. C. B. NANCREDE.

The exhibiter said that the chief point of interest was the cause of death, which had resulted neither from the original disease nor from the operation itself, but from tubercular brain-trouble induced by the presence of a caseous focus, aided by an exhausting disease. He had removed the specimens last February from a delicate little girl, aged about 4 years, an inmate of St. Christopher's Hospital for Children. So-called pathological luxation had occurred, accompanied by abscesses, sinuses, etc. The child, as is so common, seemed to have received a new lease of life consequent upon the removal of the diseased bone, gaining flesh and strength for some two weeks, when the appearance

of the wound altered, and the child's temper and general condition became very variable, until well-marked meningitic symptoms set in, terminating life nearly five weeks after the operation.

May 25th, 1882.

5. *Specimens from an excision of the hip-joint.*

Presented by Dr. C. B. NANCREDE.

These were removed in the middle of May of this year, from a girl *æt. circa* 6½ years, a patient in St. Christopher's Hospital for Children. She had suffered from the disease for over two years, had been apparently nearly cured, but had relapsed, when a large abscess of the thigh had formed, and had been opened. Great exhaustion ensued, with profuse diarrhœa and hectic, followed by the pointing of a large intra-pelvic abscess, which opened spontaneously three days before the operation. The case was a remarkably unpromising one, the acetabulum being perforated, and immense purulent collections in the thigh communicated with the intra-pelvic abscess. Notwithstanding, the patient rapidly improved, and when last seen (some weeks after the presentation of the original notes) both the operation wound and that left by the sloughing of the skin over the pelvic abscess had nearly closed.

June 8th, 1882.

6. *Chondroma of the skull.*

Exhibited by Dr. J. P. CROZER GRIFFITH.

The patient, aged 29, had been admitted to the Presbyterian Hospital, Philadelphia, June 12th. He was and had been suffering from persistent and very severe frontal headache, and held his head thrown back upon his shoulders to the farthest extent. Examination revealed a tumor the size of a walnut upon the anterior portion of the vault of the skull. This had been recognized by the patient for from one and one-half to three and one-half months; just how long was uncertain, as his headaches had very much confused his memory. He had suffered from headaches for twelve years. Ophthalmoscopic examination revealed double choked disks. While in the house the tumor grew with great rapidity, and a new growth sprang up beside it. The larger one

became soft and tender in spots. The patient became delirious, but coma and paralysis developed only three days before death, nineteen days after admission.

The post-mortem examination revealed a large tumor upon the anterior portion of the vault, consisting of a soft, red mass, but full of bone spicules, and to the right and connected with it a smaller, dense, hard, white growth. Apparently distinct capsules inclosed and separated the two. The periosteum covered both tumors. The entire growth was irregularly oval, four and one-half inches long by four to five broad, and with one and one-half inches for its greatest thickness. Removal of the calvaria showed that the greater part of the tumor was internal. Here it was divided into three lobules, of which two resembled the larger tumor in structure, while the third was more like the smaller external growth. The dura mater covered the internal projection. At points it was attached to the brain, and bone spicules penetrated into the brain substance. The length of the entire internal growth was four and one-half inches, width four inches, and the greatest depression of the brain two inches. The tables of the skull within the tumor were destroyed, and the bones around the edge eroded for some distance. The brain structure did not appear to be altered. Microscopical examination showed it to be a reticular chondroma, with a large amount of imperfectly fibrillated and rather translucent connective tissue, forming small meshes in which were cells, some embryonic, but very many cartilage cells. The bone spicules were evidently newly formed, and the older tumor had evidently undergone an ossifying metamorphosis, and then had become vascularized, forming a soft mass in which the spicules were imbedded.

Clinically the case was of interest from the existence of so great a depression of the brain with so few signs of disturbance of its functions.

March 22d, 1883.

7. *Tympanic caries producing sub-dural opening into the lateral sinus.*

Presented by Dr. W. G. DAVIS.

Neil D., aged 18, was admitted into Professor Agnew's ward in the Pennsylvania Hospital with the following history: When he was five years of age he had measles, and since that time he had had suppuration of the right middle ear. It discharged at intervals. Two weeks

previous to admission he had acute pain in his right ear, and a week later it began discharging. He had severe headache ever since he was first attacked. The pain was situated principally towards the front, but existed also towards the sides of the head. The discharge was profuse and offensive; he was emaciated; his face was haggard, pale, with a slightly yellowish tinge; he had an anxious expression of countenance, and altogether looked very bad. His mother says that this change occurred entirely during the two weeks prior to admission. The ear was cleansed and astringents applied; the discharge ceased in about six days. Bromide of potash failed to relieve the headache, but morphia, hypodermically, gave temporarily relief. He held his neck stiff, with the head slightly drawn back. This increased as the disease progressed. He had nausea, and later vomited large quantities of greenish-black fluid. Three days after entrance the pains extended down the spine and into the legs and to the under side of the arms. On the fifth day after his admission the headache and pains in the lower extremities were very severe. The discharge had almost ceased. His temperature rose to 102.5° F. An ice cap afforded no relief. On the eighth day he died.

Date.	TEMPERATURE.		PULSE.	
	Morning.	Evening.	Morning.	Evening.
9 ¹	100 ⁰	103 ⁰	62	86
10	101.5	101.5	70	96
11	98.5	102.4	66	80
12	99.5	100.8	68	84
13	102	102.5	84	88
14	102	105.4	100	102
15	103.5	102.5	106	104
16	101.6	102	136	114
17	99	100	112	108

His bowels moved regularly; they were not constipated.

Post-mortem examination made twenty hours after death. Skull was very thin at all points. Dura mater apparently normal. Marked congestion of the arachnoid and pia mater, more so on the right side. No evidences of thickening or other disease. Brain normal. No excessive cerebro-spinal fluid.

Region of Right Ear.—Beneath the dura mater covering the pe-

¹ Day of entrance.

trous portion of the right temporal bone over the middle and internal ear there was an abscess about an inch long. It communicated by a small opening with the tympanum. At its opposite extremity it opened into the lateral sinus. This was filled with pus from the point of opening to within two inches of the torcular Herophili. These two inches were filled with a soft black clot. Below the point of opening to the jugular foramen there was thick pus and a partly decolorized clot much firmer than the one extending in the direction of the torcular Herophili.

Remarks.—The congestion was active, not passive. It was most marked on the brain. The large veins were not distended, and there was consequently no excess of fluid. The patient's mind was clear to the last. The most marked feature of the case was the emaciation, which was very rapid. An interesting point in connection with the case is the cause of death. It seems to have been due to the entrance of pus into the circulation; in other words, it was probably a case of true pyæmia. He had two marked symptoms of pyæmia, the sallow countenance and the rapid emaciation; besides these, he had the vomiting. He never had the slightest chill or the sudden rise and fall of temperature, both so marked in cases of pyæmia. Sweet breath and any external abscesses or lung affection were also absent. I was unable to examine the other organs.

May 10th, 1883.

II. THE DIGESTIVE APPARATUS.

1. *Carcinoma of the liver.*

Presented by Dr. J. H. MUSSER.

I am indebted to Dr. McElree for the specimen I present to-night. The patient was attended by him during her life, and both during life and at the autopsy I had the opportunity of studying her case.

The patient was an Irish laboring woman, 58 years of age, of good habits. She suffered during the two years previous to her death. Attacks of dyspepsia, accompanied by acidity, flatulence, and irregular bowels, with uneasy sensations in the hepatic region, annoyed her during the first year of her illness. The following year the dyspeptic symptoms continued, and pain became a prominent symptom. The pain was of a dull character, irregular, however, as to severity—at one time in the hepatic region, again in the shoulders, or in the loins. The general health began to fail. In July, 1880, she had an attack of thermic fever. Three or four weeks afterwards she became jaundiced. The icterus came on gradually, and was accompanied with the usual dyspeptic symptoms and constipation.

In October, 1880, Dr. McElree saw her for the first time. I learn from him that she was much emaciated, with a dry, harsh, deeply jaundiced skin, and yellow conjunctivæ and mucous membranes. Her appetite was lost; dyspepsia continued, bowels constipated, clay-colored stools. The liver was enlarged, outline not detected on palpation; pain on deep pressure in region of gall-bladder. Heart and lungs normal.

I saw her with Dr. McElree, November 28, 1880. The jaundice had improved. Other symptoms as above noted. Pain was greatest in the lower part of back. The past ten days has had ascites. Two days ago œdema of feet began. Liver-dulness commenced anteriorly at fourth interspace; in axilla at fifth rib; lower border in both regions at margin of ribs. Left lobe seemed enlarged. Nothing noted

on palpation. Spleen slightly enlarged. Urine contained bile-pigment and a trace of albumen.

We learned that a sister had died of cancer. By exclusion, cancer of the liver was diagnosed. She died of exhaustion in ten days.

Autopsy, eight hours after death.—Rigor mortis well marked. Body emaciated; skin of a dark yellow color; œdema of feet; great distension of abdomen; tissues stained by bile.

The abdominal cavity contained about four quarts of serum. The organs were in their normal relations, and all healthy save the liver. Pushed up by the fluid in the cavity, that organ occupied the position indicated by percussion during life. Viewing it *in situ*, the gall-bladder was noted to be in front of the liver, between the right and left lobes, and two inches from the lower margin. On closer examination, it was found that the left lobe was two inches wider than it generally is; that the right lobe was irregular in shape. On the upper surface, near the longitudinal fissure, was a marked prominence, section of which showed it to be caused by the growth of a large cancerous mass, nearly the size of a base-ball, in the liver-tissue, to the left of the centre of the right lobe. There were two other cancerous masses. One was in the lower part of the left lobe, near the longitudinal fissure; the other was in a corresponding position in the right lobe, just over the normal position of the gall-bladder. Both masses had the appearance and character of scirrhus, being gray, dense, fibrous-looking structures, with an umbilicated depression on the surface. On section, they creaked under the knife; the mass was white, with yellow streaks running through; the centre was soft. The mass in the right lobe was not as distinctly circumscribed as the other, but encroached upon and extended along the left edge of the right lobe. By its contraction the curious malposition of the gall-bladder was produced. It was twisted through the longitudinal fissure and thrown on the upper surface of the liver, resting partly on both lobes. The ducts were pervious to a fine probe; the bladder was partially distended with bile. The lobules were greenish-colored in the centre, surrounded by a yellowish-white ring. On microscopical examination, the masses were of the nature of scirrhus, with degeneration of the central portions. The liver was fatty.

September 22d, 1881.

2. *Carcinoma of the pancreas, duodenum, and mesenteric glands ;
Secondary lymphatic deposits ; pericardial adhesions.*

Exhibited by Dr. J. H. MUSSER.

That the patient from whom the specimens before you were taken was suffering from cancer of internal organs was quite evident during his life. The exact localization of the deposits was not so easily determined. It was decided, however, that the disease involved the stomach, the mesenteric glands, the pancreas, the lymphatics of the neck and axilla of the left side, and probably the lungs.

The age of the patient (52 years), the rapid and extreme emaciation and loss of strength, the duration of the illness, the absence of high temperature, the occurrence of growths within the abdomen, with secondary lymphatic enlargement, although there was an absence of cancer-predisposition, pointed clearly to malignant disease. The patient was sick for three years. Dyspepsia, with poor appetite, epigastric fulness, flatulence, and acidity, dated from the beginning. Only six months prior to these observations did he fail in strength and lose flesh, and for a year previous did he suffer from pain in the back and the hypochondriac and epigastric regions.

We noted, on the 7th of June, 1881, that there was fulness of the epigastric and right hypochondriac regions and the upper half of the umbilical region, with marked bulging of the lower half of the latter region extending to the left towards the flank. An impulse was plainly seen in these areas. The abdominal veins were not enlarged. The surface-temperature was not raised. The epigastric and hypochondriac areas were hard and tender on palpation, but there was no defined tumor. The umbilical prominence extended transversely from an inch to the right of the median line, two inches to the left, and from the umbilicus to a point an inch below. It seemed to be a continuation of the upper prominence ; but on percussion there was a distinct tympanitic note between the dulness of the upper and the flatness of the lower mass. Along the course of the aorta a slight systolic murmur was noted. The gastric symptoms and the evidences revealed by palpation and percussion led us to diagnose cancer of the stomach. The separation of the lower mass from the stomach, as shown by percussion, the position and size of it, the pressure on the aorta, and the œdema of the extremities, with extreme emaciation without obstruction in the

intestinal tract, pointed to involvement of the mesenteric and lumbar lymphatic glands. During the time he was under observation he was fed on cream, and the feces examined microscopically. They were found to contain large amounts of fat, along with epithelium, granular matter, cholesterin crystals, and food *débris*. On account of the fatty stools the pancreas was thought to be involved. The post-mortem partially confirmed our ideas of the condition of the abdomen.

Inspection of the abdomen showed that the physical signs above detailed were due, in the epigastric and right hypochondriac region, to disease of the duodenum and pancreas; in the umbilical region, to mesenteric disease. The abdominal contents were matted together. The stomach was dilated and congested. Four inches of the duodenum beyond the pylorus was dilated so that its cavity would contain a base-ball. The walls were thickened, the internal surface ragged and ulcerated. The lower surface was adherent to the transverse colon, and ulceration caused a communication at this point. The omentum was not diseased. The mesenteric glands were all enormously enlarged, some being three inches in diameter, were white, of firm consistence, and softened in the centres. The lumbar lymphatics were enlarged, matted together, and adherent to the aorta, lessening its calibre. The pancreas was also the seat of disease. The large intestine was fixed between the duodenal mass and the mesenteric mass, and caused the tympanitic note at that point. The duodenal ulceration showed the fallacy of our diagnosis of pancreatic disease by the fatty stools, and explained the diarrhœa. The liver was very fatty, and weighed four pounds eight ounces.

It was further noted that the glands of the lower part of the left side of the neck were enlarged and firm, as were also those of the axilla. From their pressure the left arm was œdematous. The veins over the inner portion of the shoulder and the upper portion of the anterior surface of the chest were greatly enlarged. There was marked fulness of the first and second intercostal spaces, distinct pulsation of the subclavian, transmitting a distinct impulse to the thorax from the clavicle to the third rib. Nothing abnormal on auscultation and percussion. This negative evidence caused us to exclude lung involvement, although the fulness and impulse with lymphatic involvement caused us to think it probable. After death the lungs were found healthy above; at the left base a nodule of disease an inch square was found.

The other clinical facts of the case, which are of some interest, are as follows :—

The patient had had frequent attacks of inflammatory rheumatism, was a hard drinker, exposed to great hardship, and had much care and trouble. Inspection of the heart showed retracted impulse in the fifth interspace inside of the nipple and in the epigastrium. The percussion area was normal, the lungs encroaching properly. On auscultation at the xiphoid cartilage, a rough, high-pitched, short systolic murmur was heard. On account of the other interesting phenomena, the condition of the heart was neglected, and only the above noted. At the autopsy there were found pericardial adhesions of the anterior surface of the heart with the parietal pericardium. There was no valve-lesion.

The patient remained under observation ten days, when death took place from exhaustion, hastened by an uncontrollable diarrhoea. During life the blood was examined by Dr. Cathcart, and 2,255,000 red cells were found in a cubic millimetre, and 1 white to 225 red.

Sections for microscopical examination were made by Dr. Dunn. Both the duodenal and glandular deposits were characteristic of scirrhus.

December 8th, 1881.

3. *Fibrous stricture of the duodenum; unusual tendency to the development of connective tissue throughout the body.*

Exhibited by Dr. J. H. MUSSER.

The facts of the case briefly are as follows:—

A female; 55 years of age; married; living a life of great mental anxiety and distress, having had a large family and a cruel, worthless husband; of good habits; until the present illness had always been in good health. I attended the patient from the 23d of March, 1881, until July 11 of the same year. Early in the spring of the previous year she became weak and delicate, lost flesh, had no energy, and lost her appetite. During the summer and fall she remained about the same. During the winter she suffered from a weight and fulness in the stomach, flatulence and acidity, and constipation. Lost flesh and strength, and became sallow and anæmic. Six weeks prior to my attendance, vomiting began and continued. Dr. Girvin saw her a week before I did, and placed her on bismuth. subnit. and lacto-peptine, and a milk diet. The vomiting always occurred several hours after meals, and the ejecta were composed of a dark, thin, sour-smelling fluid, and the food eaten. Under the above treatment, the vomiting

ceased, and the patient gained flesh and strength. There were no evidences of a tumor, and, on account of the improvement, cancer was excluded. The general improvement continued during April, but she never regained her appetite. The tongue had a peculiar appearance, such as I had never seen; it was perfectly clean and smooth,—looked as if it had been shaven, apparently being denuded of epithelium and papillæ. In May she suffered from fever of an intermittent character, daily paroxysms preceded by chills or chilliness. Quinia would prevent a paroxysm partially, but did not cure them, and, on account of gastric symptoms, was stopped. The fever disappeared apparently without treatment. During this month the vomiting returned, and again she lost flesh and strength. June 1, I detected a tumor to the right of and a little above the umbilicus. It was about an inch broad and three inches in length, extending, as deep palpation showed, across the vertebral column. It was firm and slightly movable, raised by the aortic pulsation. There was no thrill, nor any pain. Temperature of abdomen was not increased; skin remained in folds when pinched; abdomen slightly tympanitic. The vomiting and constipation continued during the month, and on the 18th black vomit occurred. About this time she passed by stool a hollow cylinder, a cast of the intestine about six inches long, composed of mucus and epithelium. As the disease progressed, the abdomen became scaphoid, and the tumor was readily made out, while in the left hypochondriac and lumbar regions the dilated stomach, filled with food or gas, could be readily detected. From the persistent vomiting, the character of the ejecta, and the constipation, it was evident there was obstruction.

These symptoms continued, thrush developed along the intestinal tract, exhaustion became greater, and she died July 11 of starvation.

Dr. W. E. Hughes made the post-mortem for me. Examination eighteen hours after death. I noted as follows:—

Rigor mortis well marked. Extremely emaciated. On opening abdomen, the organs were found in their normal position. The omentum was contracted to the width of two inches, very much thickened, but there were no nodules in it, and it was almost devoid of fat. It was mostly composed of fibrous tissue, bundles of which could be readily seen. In its meshes some fat was deposited. The stomach was dilated. Three inches of the pyloric end of the duodenum was found to be of a pearl-white color, shiny, and very firm to the touch. Longitudinal section proved this to be due to a growth which so encroached upon the calibre of the gut as to cause almost complete

obstruction an inch and a half from the pylorus, a fine probe only being allowed to pass. The disease extended to, and for an inch invaded, the stomach-walls. The mucous membrane was congested and bathed with mucus. It was not ulcerated. On transverse section of the gut, four layers of tissue were readily discerned with the naked eye; a layer of the peritoneum, opaque and thickened; next, a yellowish-white layer, appearing as if made up of parallel vertical columns, and being continuous with the muscular layer of the stomach, which was hypertrophied towards the pylorus. The third layer was of a fibrous appearance, pearly white, and very dense. Where the walls were thickest, the mucous membrane of the duodenum could not be distinguished from the diseased layer; in fact, it was replaced by the mass, the membrane running up to and merging into it on either side. Over the remainder of the mass and the gut the mucous membrane was normal, and was the fourth layer. Little columns or striæ could be seen extending from the third through the second yellow layer to the first or peritoneal layer, and under the peritoneum there were several little masses. The heart was normal, and unusually small; the aorta of small calibre, with a few spots of atheroma. At the apex of the left lung a nodule of fibroid inflammation was found. That lung was bound down by recent pleural adhesions. The kidneys were small, firm, with a slightly adherent capsule and a narrowed cortical portion.

On microscopical examination, the duodenal disease was found to be due to a hypertrophy of the submucous connective tissue. The omentum was made up of an increase of the same tissue. The lung-nodule was composed of connective tissue; the kidneys were cirrhotic. It is of extreme interest to note in this case the general tendency to the proliferation of connective tissue, and also the peculiar change in the omentum. The pulmonary, pleural, duodenal, and renal proliferation can be imagined to be due to more or less long-continued irritation; the cause of the change in the omentum is without explanation, to my mind. From the observations of Bowditch and others, the size of the heart and aorta would have precluded a diagnosis of carcinoma, even without microscopical examination of the duodenum.

Dr. F. P. Henry remarked that a point of decided interest in the specimens presented was the coincidence of contracted kidneys with a small non-hypertrophied heart. On a previous occasion Dr. Henry had presented to the Society a specimen of extremely contracted kidney from a case of phthisis, in which the heart was small and flabby,

and had observed that for the production of cardiac hypertrophy an obstruction to the circulation was alone insufficient, but that, in addition, a fair state of general nutrition was essential. The case just reported illustrated this point with still greater emphasis.

In regard to the diagnosis of cancer when the physical signs of its presence are obscure, Dr. Henry remarked that he had made some observations with the hæmacytometer which convinced him that this instrument might be of decided value in this connection. In several cases of extreme cancerous cachexia he had found that the number of blood-cells per cubic millimetre ranged between two and three millions, an amount far in excess of what he had found in cases of pernicious anæmia. To quote his own words, used elsewhere in connection with the subject of blood-cell counting, "As every febrile disease has its temperature range, so the different forms of anæmia are distinguished by the degree of oligocythæmia to which they give rise."

Dec. 8th, 1881.

4. *Tumor of the spleen.*

Exhibited by Dr. JAMES TYSON.

The specimen was apparently a *gummy* tumor, involving a central segment of the spleen all the way across the shorter diameter, being two inches in its longer diameter, which coincided with the shorter one of the spleen, and one and one-half inch in its vertical or shorter diameter. It occupied the exact central segment of the spleen, which it divided into two nearly equal parts, the connective tissue of the growth being continuous with the trabecular tissue of the spleen. The spleen itself, including this intermediate new growth, was rather smaller than usual, measuring three and one-half by two inches.

The condition was unexpectedly found at an autopsy made before the class at the University.

December 22d, 1881.

5. *Abscess-sac attached to the fang of a tooth.*

Exhibited by Dr. J. B. ROBERTS.

There was no special history with this specimen, other than that of impending alveolar abscess, which had caused the sacrifice of the tooth. It well exhibited the early condition and starting-point of ordinary alveolar abscess.

January 12th, 1882.

6. *Cirrhosis of the liver.*

Exhibited by Dr. NEFF.

There was no history accompanying this specimen, Dr. Neff said, beyond that it had been removed from the body of an elderly inebriate. What had induced him to present it was the peculiar appearance presented of bright-yellow elevated spots, produced, as suggested by Dr. Seiler, by the pressure of congestion forcing out the fattily-degenerated cells of the organ. There was also stenosis of the aortic orifice, with some thickening of the mitral valves.

Dr. Tyson remarked on the peculiarity of the changes in the heart—viz., the extent of the stenosis of the aortic orifice, as such change is usually more extensive at the mitral orifice. *March 19th, 1882.*

7. *Specimens from a case of typhoid fever ; non-typhoid ulcer ; congenital stricture of colon ; gastritis.*

Presented by Dr. J. H. MUSSER.

I am indebted to Dr. Judd for the opportunity of exhibiting these specimens. I am not familiar with the clinical details of the case. The patient was a middle-aged man, a good liver, and a victim of malaria for some years. Throughout the course of the fever he suffered from gastritis, with pain in the epigastrium, great thirst, and obstinate vomiting. As early as the twelfth day he had a hemorrhage. The bowels were constipated, to relieve which an enema was ordered. After about a pint of the fluid was injected, the patient complained of such very severe pain in the left inguinal region that its administration had to be stopped. Death took place from exhaustion on the thirtieth day. Otherwise the course was typical.

By Dr. Judd's request, I made the post-mortem, eighteen hours after death. The body was not much emaciated; rigor mortis was marked. All the organs occupied their normal relations. The heart was soft and flabby, and filled with ante- and post-mortem clots. The lungs were congested posteriorly. The liver was enlarged, congested, and of a mahogany color. The spleen was enlarged, dark-red, and soft. The mucous membrane of the stomach was intensely congested, especially towards the pylorus; there were numerous ecchymoses, and some slight hemorrhages. In the small intestines Peyer's patches

were enlarged, but only a few of them ulcerated. About the middle of the ileum there was a round, punched-out ulcer, one-fourth inch in diameter, not connected with any gland, with a small clot of blood in the bottom of it, and which evidently was the source of the early hemorrhage. This ulcer was unlike any belonging to typhoid lesions, and was in existence prior to the disease. In the sigmoid flexure there was a marked congenital stricture of the gut, the lumen of which at this point only admitted my forefinger. The constriction was an inch long. The wall of that portion was four times as thick as the walls of the colon are; it was opaque and lined with mucous membrane. The enemata were evidently obstructed at this point, and hence the pain. I am unable to note the action of the bowels in health.

March 23d, 1882.

8. *Chronic follicular enteritis and colitis.*

Presented by Dr. J. H. MUSSER.

W. L., æt. 18 months, family history good, hygienic surroundings good. Under the care of Dr. Judd, to whom I am indebted for the clinical notes. July and August of 1881, he had entero-colitis, followed by a marasmic state, from which he was not restored until November. During the winter he throve well and became quite stout, although his bowels were always irregular, constipation alternating with attacks of diarrhoea. About nine days before death he had a fall, striking his head. Six days before death he had a convulsion, repeated in a few hours. The next day an acute entero-colitis set in, with the especial symptoms of tenesmus and frequent, small, bloody, and slimy stools, with fever, but without pain or abdominal tenderness. There was not much exhaustion, and death took place in a convulsion; convulsions recurred and stupor began two days before death.

I made the post-mortem for Dr. Judd twenty-four hours after death. The abdominal organs alone were examined. The body was well nourished, and rigor mortis well marked. The omentum had a normal amount of fat about it. The mesenteric and lumbar lymphatic glands were very much enlarged. The peritoneum was healthy. The mucous membrane of the stomach was congested, and near the cardiac end there was a large area of superficial erosion, evidently due to post-mortem digestion. Numerous patches of redness were seen on the

peritoneal surface. The mucous membrane of the small intestine was congested at innumerable points, with some ecchymoses. It was softened. Peyer's patches and the solitary glands were enlarged. The solitary glands were very distinct, many projecting from the surface of the mucous membrane; their orifices were enlarged and swollen, and distinguished by a black point in the dull-white colored gland. The patches were red, soft, thickened, and swollen, and appeared as if ulcerated. In the large intestine, the mucous and submucous coats were thickened and indurated; portions were congested, while other parts were very œdematous, and of an ashen-gray hue; the glands were much enlarged, and readily showed underminings of their edges, due to an ulcerative process, by the insertion of a small probe into their orifice.

The short duration of the illness, and the absence of any symptom or symptoms of entero-colitis sufficiently severe to cause death, lead me to infer that death took place from some cerebral trouble. There were no distinct symptoms of meningitis, acute or tubercular; but the fall, the convulsions, the recurrence of convulsion, etc., after the slight exhaustion from the bowel-complaint, seem to confirm such an idea, and in all probability death was caused by a clot or effusion. It is unfortunate that the brain could not be examined.

There is no doubt in my mind that some of the lesions existed since the acute disease the previous summer, notably the changes in the glands. I think it can be set down as a case of chronic disease becoming acute, and that there were present lesions common to both.

March 31st, 1882.

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9. *Cirrhosis of the liver; enlargement and dropsy of the gall-bladder; closure of the gall-duct; enlarged, pulsating, superficial epigastric vein; enlarged spleen; ascites, hydrothorax, œdema of the abdominal walls and of the legs; absence of early dyspeptic symptoms and of spirit-drinking; cause, possibly chronic lead-poisoning. Autopsy.*

Presented by Dr. J. H. MUSSER.

On the 11th of November, 1881, Mr. S. applied to the medical dispensary of the Hospital of the University for treatment. He was 41 years of age, a car-painter, of good habits, and free from the suspicion of hereditary taint or specific disease. He was married; the father

of healthy children. He stated that his illness was of a duration of one year, and that it began with slight jaundice, which gradually increased in intensity. He did not remember any dyspeptic symptoms previous to or since the jaundice. His abdomen began to enlarge three or four months previous to the visit. His bowels had always been constipated. He had lost flesh and strength.

We noted then as follows: features sunken; marked venous stigmata on cheeks and nose; dark-yellow hue of skin, and yellow conjunctivæ; emaciation; harsh and dry skin; distension of the abdomen; œdema of the feet. As noted, the abdomen was considerably enlarged, and, on palpation and percussion, fluid was detected in the cavity. The superficial veins of the right side were not enlarged, but the superficial epigastric vein of the left side was very much enlarged, tortuous and pulsating, and midway between the umbilicus and the xiphoid cartilage, an inch from the median line, it dipped down through the abdominal walls.

The appetite was good; tongue clean and red; no dyspeptic symptoms; constipation; stools yellow or brown; no hemorrhoids. The liver-dulness was lessened, extending in the nipple-line on deep percussion, from the fifth rib; on light, from the sixth interspace to one inch above the margin of the ribs. In the axilla the dulness was three inches in width. Palpation negative. The spleen was not enlarged. Urine the color of porter, frothy, and showed, with nitric acid, bile-pigment. There was a slight trace of albumen and uric acid; the amorphous urates were abundant. No casts or epithelium. Treatment, diuretic mixture containing tinct. digitalis, potas. acet., acet. scillæ., spt. juniper., comp. liq. ammon. acet., and a draught containing 10 grs. ammon. mur., to the dose, three times daily.

He continued his visits to the dispensary till the 29th of the month, all the while improving, so that on the last visit he had scarcely any ascites, no œdema of the feet, and much clearing up of the skin. Called to see him January 5, 1882. Found him in about the same condition as on his first visit, save that he had in addition severe cough and dyspnœa, caused by a right hydrothorax, and a hydrocele, and much flatulence, causing suffering. Under the treatment detailed below, the œdema of the feet and the hydrothorax subsided; the ascites diminished. The wasting, however, progressed, the features became more sunken, the face more injected, the jaundice continued. The tongue remained red and raw; flatulence and a "stuffed-up" feeling mostly annoyed him. The bowels were loose. The urine, which had been

scanty, increased in amount. Its characteristics were the same as when first analyzed.¹

To show that there was serious internal obstruction to the circulation, there developed at first in the right hypochondriac and the lower half of the lumbar and umbilical regions capillary injections in half-moon patches; the entire skin of the abdomen in the later stages was so marked. The spleen was enlarged to twice its natural size.

Treatment.—On the first visit rest and milk diet, ammon. mur. as before, and a mixture of acet. potas. and inf. digitalis was prescribed. The latter nauseated him so that its use had to be abandoned. Citrate of caffein was of good service, increasing the flow of urine perceptibly; it caused wakefulness, however, so that chloral had to be added, and the combination did not act so well. Digitalis, calomel, and squill in pill were of some service, but soon so nauseated him that they had to be given up. After the dropsy and diminished supply of urine, flatulence was the most serious symptom. Carbolic acid, bismuth, charcoal, pepsin, etc., singly or in combination, were of no avail, while spirits of turpentine not only gave prompt relief to the flatulence, but acted well as a diuretic and vascular stimulant. With caffein and turpentine, rest, and restricted diet, he was brought to the condition mentioned above. Anasarca much diminished. Paracentesis was frequently urged, and as frequently refused.

February 8. His condition remains at about a stand-still. At a consultation, calomel one-tenth grain, pulverized ipecac one-twelfth grain, in pill, every three hours, were ordered. Sodæ et pot. tart. 3j, and ac. tartaric. 3j, in Oj of water, to be taken in twenty-four hours. Again increased diuresis for a short time, with relief, occurred, to be followed by an aggravation of symptoms. Elaterium and calomel *pro re nata* replaced the above pill. Hot air and Turkish baths were tried, with no avail. Jaborandi caused diaphoresis, and relieved anasarca considerably.

From March 2d to day of death, he was attended by another physician. Anasarca increased, jaundice became more intense, urine diminished, slight capillary hemorrhages occurred in the mouth and throat. The last two months, he complained of pain in the epigastrium and hypochondrium, and the twenty-four hours previous to death, of excruciating pain in the lower part of the abdomen. Death

¹ The notes as to the amount of urine passed daily under various forms of treatment were mislaid, as well as the various measurements of the abdomen and feet.

took place from exhaustion, mind clear, on March 24. For six weeks previous to death a characteristic feature was œdema of the abdominal walls and back.

Forty hours after death I made the autopsy. Rigor mortis well marked. CEdema of feet, of abdominal walls, and back. Skin discolored from jaundice; ecchymoses on back. Abdominal cavity contained three gallons of serum. No peritonitis. Dissection of the superficial epigastric vein showed that at the point mentioned in the clinical notes it dipped down through the abdominal wall, and ran along underneath the muscular fascia, between it and the peritoneum, to the falciform or suspensory ligament of the liver, in which it anastomosed with enlarged veins. One of these veins, as shown by Sappey (quoted by Trousseau), "enters the left branch of the portal sinus, where it is attached to the cord of the umbilical vein." There was no peritonitis. Liver weighed thirty-two ounces; right lobe measured three inches transversely, and four antero-posteriorly, the left lobe two and one-half, and four inches respectively. It was a perfect example of true cirrhosis. The gall-bladder was three times its natural size, distended with clear serum. Its mucous membrane was swollen, roughened, and congested. It contained no gall-stones. The gall-duct was impervious; the hepatic and common ducts were much dilated and inflamed. The kidneys were enlarged, hard, and congested. The spleen was enlarged to twice its normal size, dark-red, firm on pressure; its capsule thickened. The right pleural sac contained a large amount of liquid. The heart was soft and flabby and bile-stained. All the tissues were stained with bile.

It is of importance to note in the case the absence of spirit-drinking as an etiological factor. I am disposed to think the exposure to lead in following his occupation, although other lead-symptoms were absent, was the cause of the cirrhosis. If it be true, it shows distinctly that the early dyspeptic symptoms of cirrhosis, so markedly absent in this case, are due to the local action of the irritant alcohol, for the poison in the case was inhaled, and hence there was no irritant to excite dyspepsia. On account of the absence of alcoholism and dyspepsia, the diagnosis was somewhat difficult, and for a time carcinoma of the liver was considered. The diminution in size, and the enlargement of the spleen, were strong factors. The most important point to us was the enlarged external vein. I have never seen or heard of its occurrence to so marked a degree in cancer of the liver. The increased size of the gall-bladder was not made out during life, possibly

because of the tense and afterwards œdematous abdominal walls, and partly because of the organ having been behind the ribs and higher up than normal.

I cannot account for the condition of the gall-bladder and duct, either by symptoms or by the post-mortem appearances. The patient never, to his knowledge, had calculi or any severe local inflammation. It may have been a congenital state. There is no doubt that the common and hepatic ducts were inflamed and aided in the causation of the jaundice. Early and frequent tapping, I believe, would have prolonged life, had it been permitted by the patient.

May 11th, 1882.

10. *Sclerosis of the head of the pancreas.*

Presented by Dr. J. Trson.

The specimen was derived from a lady aged 68, who, while for years delicate in appearance, had very little serious illness until February, 1880, when she had a very severe attack of catarrhal pneumonia which involved successively both lungs. For a year or two before and since that time she would have occasional attacks of flatulent colic with constipation, apt to terminate in diarrhœa. In July, 1881, while residing for the summer in a most healthy mountain district, she was seized with a diarrhœa in which the stools were typically clay-colored or ashen in appearance. There was uneven and irregular distension of the abdomen, as though circumscribed areas of bowel were distended with gas. I visited her in July, 1881, and found such a lump, which seemed rather more than usually fixed, just to the left of and below the umbilicus. She only partially recovered from this diarrhœa, and remained very weak. She returned to the city in October, 1881, feeble and emaciated, with a distinct tumor just below and to the left of the umbilicus. A part of this lump was evidently gas, but at its base I thought I could detect a harder and more resisting portion, which was also tender on deep pressure. But of the presence of this hard base I could not then be certain. The diarrhœa continued, and all attempts completely to control it failed. For two or three days at a time, under the use of opiates and astringents, there would be no movement, when would come a sense of discomfort which was relieved by a discharge, at first formed, but finally liquid. The stools soon became distinctly

fatty, the milk which formed her almost exclusive diet seeming to be discharged little altered, although there was often some semi-liquid or even partly-formed ashen-hued fecal matter. The same knots of distended intestine continued present at various situations and with varying degrees of distinctness. As she grew thinner, the lump in the neighborhood of the umbilicus became more and more distinct, until I was satisfied that it was a tumor above which lay a knuckle of intestine distended with gas. Hard percussion could always bring out dulness, and the tumor was very sensitive and tender. The fatty diarrhœa continued, and she grew weaker and thinner and more bloodless, until she seemed a mere shadow of her former self, and the abdominal organs could be easily mapped out through the thin walls.

There was never any vomiting, although there was a good deal of nausea at times. Her circulation and respiration were unaffected. The diagnosis of pancreatic disease, rather than of cancer of the stomach, which was made before death, was based upon the absence of vomiting and the persistent fatty diarrhœa. She died March 13, 1882.

The autopsy was made twenty-four hours after death. The lump near the umbilicus, easily visible before death, had almost entirely disappeared, in consequence of a uniform distension of the abdomen. There were no lesions of the heart, lungs, stomach, or liver, but about the head of the pancreas were a number of adhesions, so that it was with considerable difficulty that the head of the organ could be isolated. The pancreas was removed with a portion of the gut attached, and found to be hard and resisting, but little enlarged.

Dr. O'Hara thought that the fatty stools as diagnostic of carcinoma of the pancreas, to say the least, were unreliable.

Dr. Tyson thought, on the contrary, that the early appearance of this symptom, and its long persistence, as in this case—viz., nine months—were of the utmost value in deciding upon the presence of pancreatic disease.

Dr. Shakespeare said that Dr. Tyson's case recalled one with similar symptoms, the history of which Dr. Curtin read at the last meeting of the State Medical Society. It presented many similarities, such as fatty diarrhœa, etc., and the diagnosis made was carcinoma of the pancreas. At the post-mortem examination the morbid appearances presented were similar to those seen in Dr. Tyson's specimen. Upon microscopic examination by him, no evidence of malignant disease was found, the pathological changes consisting chiefly of increase

in the periglandular connective tissue, and catarrh of the ducts and acini. As he remembers it, the gist of Dr. Curtin's paper was the claim that the disease was really a catarrhal pancreatitis.

Dr. Musser asked if there were any bloody stools, as such have been described as constantly occurring in chronic pancreatic affections, such as sclerosis, pancreatic calculi, etc.

Dr. Tyson replied that it was interesting to note that the family had referred to this symptom, having told him that occasional bloody stools had been observed. He had not seen them himself, and was therefore in doubt, knowing the tendency of patients to describe discolored stools as bloody. The patient had been fed on milk. The tumor was much smaller and less characteristic in appearance than when removed. The reagents had changed the appearance of the specimen.

Dr. Nancrede thought that the specimen was unlike any case of pancreatic carcinoma he had observed, in that after so long a time the growth was so small; and he was inclined to think, in default of microscopic examination, that it was of a benign nature, death resulting from exhaustion owing to the non-absorption of fatty food from deficiency of the pancreatic secretion.

May 11th, 1882.

Report of the Committee on Morbid Growths:

"A microscopical examination of a thin section of the pancreas shows an increase of its fibrous tissue, and an atrophical condition of its secreting structure. The change may be termed a sclerosis of the organ."

June 22d, 1882.

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11. *Lesions of typhoid fever; small circumscribed sphacelus involving all the coats of the bowel in the centre of an ulcerated patch of Peyer.*

Presented by Dr. J. H. MUSSER.

The specimen was derived from a young lady, who died of exhaustion at the end of the twenty-fifth day. The case was one characterized by constant high temperature,—105° Fahr. being noted in the morning of the twelfth day,—intense headache, and active delirium from the start. The temperature was 104.1° in the evening, twenty-four hours before death, and seemed to be as high almost up to the moment of death.

In the delirium there was constant talking, crying, and even screaming, to which was added, towards the close of the illness, troublesome cough, so that it was thought that there might be miliary tuberculosis and meningitis; but at the autopsy, made twenty-four hours after death, no lesions were found in the brain or lungs, not even a diminished transparency of the pia mater, nor the slightest effusion into the ventricles, while the sinuses of the dura mater appeared somewhat turgid with blood. Many of the Peyer's patches in the upper part of the ileum appeared to have nearly healed, but in the lower part were two large unhealed ulcers, in the centre of one of which was an oval, opaque, white patch, presenting the same appearance, when viewed from the peritoneal or from the mucous side. It was about four lines in its longer diameter and three lines in its shorter. On touching it with the end of the little finger, it loosened from its peripheral attachment at that point with the greatest readiness, and there can be no doubt that, had she lived twenty-four hours longer, the little sphacelated piece would have dropped out and death taken place from peritonitis.

May 25th, 1882.

12. *Rupture of the descending colon from external violence.*

Exhibited by Dr. J. EWING MEARS.

The specimen had been removed from a patient in St. Mary's Hospital. The autopsy was made by Dr. Strittmatter, resident physician, under whose care the patient was, and who furnished the following notes of the case:—

Charles F., aged 14 years and 6 months, was admitted into the surgical ward of St. Mary's Hospital, May 12, 1882. He was engaged on the third floor of a factory in carting material to and from an elevator. He was in the habit of walking backward when putting his cart on the platform of the elevator, pulling the cart after him. On this occasion, however, the elevator had ascended a story higher, and, instead of stepping on the platform, he fell down the elevator-way to the ground-floor. Whether he struck anything previous to his contact with the lower floor cannot be positively ascertained. The cart, which weighed about fifty pounds, came down with him. When the boy was first seen, the cart was lying on him. His injuries, as far as could be detected on examination, were contusions on the back

and below the right ear, fracture of the right tibia and fibula, and contusion of the abdomen.

When brought to the hospital, he was conscious, and complained of severe pain in the lower portion of the abdomen. He spoke hurriedly and as if in great agony. The surface of his body was cold, and his forehead and face were covered with cold perspiration. His countenance had a typically pinched expression. His skin presented a waxy paleness. His pulse was small, hard, 160; respiration quick, shallow, and at times sighing and gasping. Temperature, 97.5° in the axilla. Morph. sulph. gr. $\frac{1}{4}$ was administered hypodermically, and brandy in f5ij doses was given by the mouth every fifteen minutes. Hot bottles were applied to the extremities. In half an hour no improvement had taken place; the pulse grew more feeble and rapid, and respiration more sighing and rapid. Tinct. digitalis gtt. viij and atropiæ sulph. gr. $\frac{1}{80}$ were now given hypodermically. No perceptible change, however, followed this. His pulse grew more feeble, and the respirations still more rapid.

He became uncontrollable, tossed his arms wildly around him, heaved deep sighs, and constantly clamored for water.

He recognized his mother and sister, but was unable to speak to them. In about an hour he became quiet, began to sink very rapidly, and two hours after admission died.

During life no careful examination was made of his body, excepting his head and what could be obtained by careful inspection, on account of the shock, from which he was suffering profoundly. By this no injury of the brain could be discovered, and none was manifested by any symptom, excepting the vomiting which took place about one and a half hour after admission. On his abdomen in the left lumbar and inguinal region there was evidence of contusion. The muscular tissue seemed to have been separated, and allowed considerable bulging. On light percussion this gave rise to a somewhat amphoric sound. Moderately deep percussion gave dulness. Percussion on either side gave evidence of a distinct line of dulness. Fluctuation was distinct. The catheter had been passed, and about two or three ounces of clear urine came away very slowly. More could not be discovered in the abdomen. His right tibia and fibula were fractured in the middle third.

Post-mortem examination revealed the following condition of the abdomen. In the left lumbar and iliac regions the skin was discolored by extravasation of blood. It was not torn. The abdomen was dis-

tinety bulging on this side. An incision showed this to be due in part to a clot in the connective tissue between the skin and the external oblique muscle. The greater portion of this clot was found two inches internal to, and on a level with, the anterior superior spinous process of the ilium. Under this there was no separation of the muscular fibres, only a bruised, infiltrated spot.

Between the external and internal oblique there was found a quantity of coagulated blood, which was not so much circumscribed as that between the integument and the external oblique. There was also more extensive bruising of the internal than of the external oblique. Between it and the transversalis muscle everything was full of clot, while the transversalis was cut through or some of the fibres were separated a short distance internal to, and on a line with, the anterior superior spine of the ilium. The parietal layer of peritoneum was separated by an effusion of blood. The whole abdominal cavity was filled with blood. The only injury discovered in the abdominal organs was the rupture of the colon about two and a half or three inches above the sigmoid flexure.

Owing to the haste with which the autopsy was made, a careful examination of the posterior wall of the pelvis was impossible. Nothing was noticed, however, on careful removal of the ruptured bowels. A small lump of hardened feces was found in the abdomen near this rupture, and a prune-seed in the opening; several seeds were above, one or two below.

No fracture of the pelvis could be discovered on careful examination.

Between the parietal layer of peritoneum and abdominal walls, anterior and posterior, there was abundant extravasation, so that without a careful dissection it was impossible to determine the amount of injury there.

It is evident from the nature of the injury sustained in this case that the bowel was ruptured by the direct violence inflicted by one of the projecting handles of the cart, which fell to the lower floor with the patient, and beneath which his body was found. It is interesting to consider to what extent the prune-seeds which were in the intestine contributed to the laceration of its walls. It is quite probable that they assisted materially, cutting the coats of the intestine with their sharp edges, as the bowel was caught with the abdominal wall between the handle of the cart and the resisting bony wall of the pelvis. It

will be noted that one was found in the opening of the upper portion of the lacerated colon.

During the two hours of his sojourn in the hospital after the accident, the symptoms indicated, in a marked manner, the existence of internal hemorrhage, in the character of the respiration, the pulse, and the temperature.

Dr. Tyson thought that the mechanism of this rupture necessitated some more or less solid body in the bowel, and that the prune-seed was the object which afforded the necessary point of resistance, as demonstrated by the persistence at certain points of the peritoneal coat, showing that the bowel had been divided by some dividing force acting *from* the lumen of the gut *outward*.

Dr. Mears agreed substantially in the views expressed by Dr. Tyson.

May 25th, 1882.

13. *Rupture of the spleen.*

Presented by Dr. FERDINAND H. GROSS.

J. G., a heavy, muscular American, aged 38, fell from, and was run over by, an ash-cart, at noon, June 16, 1882, the wheel passing over his body from the left side about the region of the eighth rib. He was carried to the German Hospital, where he was taken charge of by my resident, Dr. L. D. Brose, who found the patient in a profuse perspiration and complaining mainly of pain in his left side. On removing the clothing, no external discoloration or visible marks of contusion were discovered; but by careful palpation the eighth and ninth ribs of the left side were found to be fractured. The right clavicle, at the junction of its sternal with its middle third, was also fractured. Dr. Brose applied adhesive strips to the left half of the chest, and for the fractured right clavicle a Velpeau bandage. During the first night the patient vomited food. I did not see him until morning. In the course of the day some pain was complained of in the right side from the Velpeau bandage, which was directed to be removed. This fracture having been adjusted by means less constricting to the chest, the patient rested somewhat easier, but his countenance still retained the expression of great anxiety and suffering, and he complained much of thirst. Cracked ice was given, and occasionally two teaspoonfuls of liquor morphiæ sulphatis were administered. His diet consisted of

milk with lime-water. In the evening of the 17th the temperature was 100.8°; pulse, 112. On the morning of the 18th the patient was reported as having vomited blood during the night; he still complained of great thirst, and was bathed in a cold sweat. The abdomen had become tympanitic and painful. Morphia and atropia were administered hypodermically, but vomiting was only partially checked by this and other remedies. When it recurred later in the day, a dark, grumous matter, which was ascertained to be altered blood, was thrown up. A passage from the bowels, not containing any blood, was effected by an enema. Indications of serious internal injury, which were suspected on the previous day, were now unmistakable, and the spleen was believed to be ruptured. The morning temperature on the 18th had lowered to 100°, while the pulse had risen to 132. The bad symptoms remained uncontrolled by remedies applied internally and externally. The patient's mind remained clear until about fifteen minutes before death, when the temperature rose to 104.4°. He died at 5 P. M.

Autopsy.—Found an extravasation around the seat of fracture of the clavicle, and a similar extravasation about the fracture of the ribs. Heart and lungs normal, except a slight congestion of the latter. In the left pleural cavity there was a small effusion of bloody fluid. The intestines were greatly distended by gas, and the abdominal cavity contained a large quantity of blood. The spleen, which is here presented, shows a large rent in its upper portion, and is heavy from infiltrated and coagulated blood. Stomach and liver normal.

June 22d, 1882.

14. *Cirrhosis of the liver in the stage of enlargement.*

Exhibited by Dr. E. T. BRUEN.

T. W., aged 20 years, colored; has worked on a farm since boyhood, and has been much exposed to weather. His habits were temperate; he was free from either syphilitic or malarial taints. Father is still alive; the mother died of phthisis. He was never robust, but had had only one severe illness, viz., typhoid fever, from which he convalesced perfectly, but he readily "took cold." He was first seen by Dr. Bruen at the University Hospital, in January, 1882, when he gave the following history:—

Abdomen began to swell two years ago, with neither pain nor tenderness. Was obliged to rise at night, to urinate. The abdominal swelling increased, and the previously regular bowels became constipated. Occasional sharp, shooting pains were felt across the chest when lifting weights or working hard, and also slight, dull pain over the liver, lasting for a few moments only. When seen the abdomen measured fifty inches. On the 19th of January he was tapped, nineteen quarts of fluid being removed, rendering plain a much enlarged liver, covered with smooth, nodular elevations, with the apex beat of the heart displaced upward into the fourth interspace. March 18th, five gallons more fluid was removed, after which pleural and bronchial complications arose, which soon subsided. May 6th, paracentesis by capillary needle was resorted to, which was followed by much localized tenderness around the site of the punctures. Symptoms of peritonitis developed next day, which terminated life the same evening. During life, the diagnosis was most difficult and interesting. The enormous size of the liver, the palpable bosselation of its surface giving a sensation like that of crepitating tissue, as though fluid-lymph had been thrown out; and finally his abstemious habits, with absence of either syphilitic or malarial taints, suggested malignant disease. His age, the excessive rarity of primary carcinoma or sarcoma of the liver, with his family history, all negatived this view. He had had some dyspepsia. Enlargement of the liver connected with catarrh of the bile ducts, would have presented symptoms of jaundice, and intermitting temperature, terminating by death from cholesteræmia. The case, then, was one of simple cirrhosis.

Sectio cadaveris.—The abdomen contained six gallons of purulent fluid. Both the parietal and visceral peritoneum were covered with a thick coating of inflammatory lymph, tinged with blood, from multiple capillary hemorrhages. The abdominal veins were all replete with blood. The liver weighed nearly $5\frac{1}{2}$ pounds, was of a nutmeg appearance; on section was indurated, and presented a bosselated, nodular appearance. The gall-bladder was thickened and contracted about two-thirds in bulk. The bile-ducts were normal. The spleen was covered by a pseudo-cartilaginous capsule but was otherwise normal, as were also the stomach, pancreas, intestines, kidneys, and suprarenal bodies. The abdominal lymphatic glands were slightly enlarged.

Dr. Seiler remarked that, having seen the case during life, it was almost impossible to divest one's self of the idea of malignant disease. He thought that the projections were the unaltered portions of the

liver, which had been compressed and squeezed out by the contracting interstitial tissue.

Dr. Bruen remarked upon the obscurity of the etiology.

Dr. Tyson asked Dr. Formad, who had examined the specimen microscopically, whether he considered it to be in the first or second stage of the affection.

Dr. Formad replied that he considered it to be in the commencing second stage, and detailed the microscopical appearances.

Dr. Tyson, after briefly adverting to the causation of cirrhosis, said that his reason for asking Dr. Formad whether he considered that the organ was in the first or second stage of cirrhosis was, that some few years since an important insurance case had been argued in our courts, where the defence was set up that the man had not a cirrhused liver, because it was enlarged. For his part, he had no doubt that a liver could be in the second stage of cirrhosis and yet be enlarged; there might be enlargement from fatty infiltration, concurrent with interstitial inflammation.

October 26th, 1882.

15. *Primary carcinoma of the pancreas and liver.*

Presented by Dr. EDWARD T. BRUEN.

The interesting features pertaining to this case are the age of the patient (24 years), and the rapidity of the abnormal processes. These rendered the diagnosis of malignant disease doubtful, until the appearance of nodular tumors in the liver.

The family history was free from hereditary disease. The commencement of the disorder dated from Sept. 1881; death occurred on the 15th of Jan. 1882.

At first, the symptoms related solely to the digestive tract, such as dull heavy sensations after eating, with acid eructations and occasional vomiting. Subsequently, sharp cramp-like pains in the abdomen were a prominent symptom. After the lapse of a week there commenced general itching; two weeks later, the skin became yellow. This yellowness and itching never disappeared from the history of the case. Vomiting and eructation of gas were troublesome symptoms. At the autopsy the gall-duct was found to be obstructed by the enlarged head of the pancreas, so that extreme dilatation of the gall-bladder had ensued; this probably explains the jaundice, and not the morbid pro-

cesses found in the liver itself. These symptoms were complained of at the time when the patient was admitted to the University Hospital, the 20th of Nov. 1881.

At this time the skin was stained a deep yellow; the general pruritus was intensely annoying. The bowels were regular, the appetite good. The ensemble presented the evidence of partial obstruction of the gall-duct, with digestive disorder, but without the symptoms characteristic of disease of the stomach or bowels.

In the diagnosis of tumor of the pancreas, the testimony negating disease in the stomach may be suggestive.

Dec. 14. Liver dullness commenced at the fourth interspace, and extended three inches below the ribs, in the nipple line, from the ensiform cartilage; the line of dullness extended to within an inch of the umbilicus. The hepatic region was tender on pressure, especially over the epigastrium.

The patient complained about this time of dull pain in the liver, with gripping pain in the abdomen; pulse was 76 per minute. Four pounds of flesh had been lost since admission, and the patient looked thin.

About this time, the 20th of December, a small inequality was noticed on the surface of the liver, three inches above, and a little to the inner side of the umbilicus; the spleen also was enlarged.

Jan. 7. Bossellation of the liver was more noticeable than ever. To the right of the epigastrium and umbilicus the enlarged gall-bladder presented a slowly increasing, elastic tumor, painful to the touch. Inequalities caused by gall-stones could be recognized.

12th. The pulse, which at first was slow, between 60 and 70 per minute, became rapid, 130 per minute; the patient quickly failed, and death occurred Jan. 15, as already stated.

An autopsy revealed a pancreas enlarged to double its physiological size, the tumor being situated at its head, and occasioning pressure on the common biliary duct.

Microscopic examination revealed its nature to be scirrhus carcinoma. The liver was thickly studded with nodules of medullary carcinoma, explaining the bossellated sensation on ante-mortem palpation. The gall-bladder was distended to twice the physiological size, and contained a number of gall-stones.

In commenting on this case, I would add that the duodenal end of the pancreas, as in this case, is the common location of disease. In St. Bartholomew's Reports for 1881, a record of thirty-nine cases of

carcinoma of the pancreas can be found; in these cases it is stated that jaundice always accompanies *primary* disease of this organ.

In secondary carcinoma, of twenty-four cases, seventeen escaped jaundice, presumably from the location of the pathological process in secondary carcinoma elsewhere than in the head of the gland.

Murchison says: "The characteristic symptoms of cancer of the pancreas are, pain in the pancreatic region, sensible tumor, and persistent jaundice." To these I would add intestinal dyspepsia, which differs in some essential particulars from the dyspepsia incident to organic disease of the stomach.

November 9th, 1882.

16. *Spindle-celled sarcoma of the small intestine.*

Presented by Dr. W. A. EDWARDS.

On September 23, 1882, I was asked to assist Dr. W. F. Atlee in the removal of an abdominal tumor. The patient, æt. 48 years, whose menstruation had ceased at 31 years, first noticed the swelling in April last; on the day of the operation she measured thirty-eight inches around the abdomen. The usual incision was made and the tumor reached, when its surface was seen to be of a dark purple hue, with a network of large veins ramifying in every part of its serous covering. A trocar and canula were introduced, but nothing but blood followed the withdrawal of the trocar. The sac was then torn open and its contents, of a soft, brain-like consistence, were emptied out. The growth was now turned out of the abdominal cavity. There was no distinct pedicle, but an attachment to the intestine, of about the size of a half dollar, was seen. Dr. Atlee says, "when I emptied the sac of its soft contents, I examined carefully, with *extreme care*, the part fastened to the intestine, and my fingers passed into the intestinal tube." A silk cord was tied around the attached portion and the remainder of the growth removed. The omentum was attached to the growth for a space of two inches; this was ligated and cut away, and the abdominal wound was closed, etc.

Death occurred on September 25, at 4 A. M.

This growth sprang primarily from the submucous tissue of the small intestine, and grew with great rapidity, as the patient was only aware of its presence last April, and by September she measured, as above stated, thirty-eight inches.

Microscopical examination of preparations taken from several por-

tions of the growth clearly show it to be a spindle-celled sarcoma, and a most typical one at that. The small intestine is an unusual site for this neoplasm. As far as I am able to ascertain, there is no recorded instance of its occurrence in this situation. My friend, Dr. Formad, to whom I have shown the growth, concurs with me in this statement. On the day of operation I noted, as well as I could, the absence of all secondary deposits. The surrounding intestines and peritoneum were apparently normal, not even unduly hyperæmic. No post-mortem was permitted.

November 9th, 1882.

17. *Ecchymosis of the mucous membrane of the stomach.*

Presented by Dr. J. M. BARTON.

The history of this case was that of chronic lung trouble. The stomach upon being opened presented an irregularly shaped extravasation of blood, about two-thirds of an inch in diameter. The mucous membrane covering the effusion was healthy, as it was in the rest of its extent.

Dr. Tyson remarked that these effusions are not uncommon, but he had never seen them except in their pin-point form.

Dr. Roberts asked if there had been violent vomiting recently.

Dr. Barton replied that nothing of this sort had been observed for some months prior to death.

November 9th, 1882.

18. *Specimens showing abscess of the liver, resulting in gangrenous inflammation of the cæcum and its appendix with sloughing of the latter, a circumscribed abscess sac in the right ilio-lumbar region, and multiple abscess of the spleen and lungs, occurring in a person suffering from severe heart lesions.*

Presented by J. T. ESKRIDGE, M.D.

Through the kindness of the other members of the medical staff of St. Mary's Hospital, I have been able to uninterruptedly pursue my investigations in surface thermometry during their terms of service at that institution. The specimens I present to-night were taken from a woman who had been a ward patient under the care of Dr.

Hickman. As her case was an interesting one, in which to test the value of surface thermometry, and as multiple lesions rendered an accurate diagnosis difficult, I made almost daily temperature observations on her, carefully noting each symptom during the two weeks immediately preceding her death. I wish to thank Dr. Hickman for the specimens, and Dr. Everett, resident physician, for the tabular view appended representing his observations, and for this courtesy and assistance in aiding me in studying the case.

Fannie, æt. 27 years, factory girl, born in Philadelphia, was admitted into St. Mary's Hospital Oct. 16, 1882. She gave the following history: Her father had complained of shortness of breath a long time, and died suddenly seven years before, while engaged at his work. Her mother and two of her sisters were well. Four sisters were dead, two died young, and the other two from heart disease. She suffered from inflammatory rheumatism when quite young, and from a second attack, which confined her to bed two weeks, five years ago. After each rheumatic seizure she felt well and as strong as usual, not being troubled with shortness of breath, or other evidences of heart disease, even on ascending long flights of stairs. Excepting intermittent fever, from daily paroxysms of which she suffered for a week, two years ago, she enjoyed very good health until Sept. 20 of the present year, when, after sitting on marble steps several hours one afternoon during her menstrual period, she was awakened about 1 A.M. of the night of the same day by cramping pains all over the abdomen. Shortly after she had a chill, and suffered from nausea and vomiting. The menstrual flow had stopped the evening before. The next morning the pain was localized in the right iliac and hepatic regions. Vomiting, attended by some fever, lasted two or three days, after which she worked about a week, when, on account of severe pain in the right side of the abdomen, dizziness, and general weakness, she was compelled to take to her bed. She was seen by a physician only twice before she was brought to the hospital. She was considerably jaundiced during the early weeks of her sickness. The first eleven days of her stay in the hospital no record of her condition was kept. Her bowels were costive the first three weeks of her illness, but afterwards they moved irregularly.

On Oct. 27 and 28, the first days that I saw her, her temperatures and general symptoms were noted to be as follows:—

27th, 11 A. M. Temperature of room, 70°. Axillary temperature,

100.3°; pulse, 100; respiration, 26. Temperature of iliac regions, right, 100.4°; left, 100.4°.

28th, 11 A. M. Temperature of room, 71°. Axillary temperature, right, 99.4°; left, 99°; pulse, 108; respiration, 30.

Temperature of iliac regions, right, 99.6°; left, 97.7°. Temp. anterior lumbar, right, 100°; left, 99.8°. Temp. posterior lumbar, right, 100.9°; left, 101.2°. Temp. lateral hypochondria, right, 100.5°; left, 100.3°. Temp. posterior hypochondria, right, 101°; left, 100.6°. Temp. axillary region of chest, 7th intercostal space, right, 100.3°; left, 100.3°. Temp. anterior surface of chest, fourth intercostal space, right, 98.8°; left, 99.3°. Temp. hypogastrium, 99.4°; umbilical region, 99.6°. She was anæmic, emaciated, and slightly jaundiced. Her features were pinched. The subcutaneous tissue of the chest appeared œdematous when pressed upon by the stethoscope. She complained of great prostration, loss of appetite, irregularly appearing chilly sensations, followed by fever, nausea, and at times vomiting, diarrhoea followed by constipation, and pain and tenderness in the right lumbar and iliac regions. The upper anterior portion of the crest of the right ilium was the most sensitive part of her body.

Physical signs connected with the heart.—There were no visible arterial pulsations, except over the carotid and subclavian arteries. Impulses were seen over the heart, in the second intercostal space on the right side, and in the second intercostal space to the sixth inclusive on the left. The lower pulsations covered a broad area, and extended from near the left border of the sternum to a perpendicular line running midway between the left nipple and anterior axillary border. No venous pulsation was seen. The pulse was moderately full, but weak; yet it was irritable, and appeared to come up to the finger with considerable force. The pulsation in the left second intercostal space seemed to be presystolic, the other præcordial pulsations systolic. A decided presystolic thrill was felt in the third, fourth, and fifth left intercostal space. Cardiac dulness increased, downward, and latterly to the left and right. Systolic murmurs were heard over the aortic, pulmonary, and mitral valves. The aortic murmur was loud, moderately rough, and transmitted with considerable intensity into the right and left carotid and subclavian arteries; the murmur in the pulmonary artery was soft and blowing, and that at the apex was tolerably distinct, decidedly rough, and plainly heard posteriorly. A very rough presystolic murmur was audible over the third and fourth left costal cartilages. The lungs were apparently healthy. During

inspiration, when the stethoscope was held near the apex of the heart, peculiar squeaking, cooing, or moist bronchial-like sounds were heard, corresponding in time and frequency with the cardiac systole. At my second examination of the heart, in regard to these sounds, I made a note which reads, "They are probably due to intra-pleuro-pericarditis, or mucous rales in the bronchial tubes, more likely to the former, as no cough or bronchitis exists, and as the sounds have remained unchanged in character and situation since yesterday."

All parts of the abdomen were tolerant of manipulation except those in and immediately around the right iliac region and lower portion of the right lumbar. Here tenderness was so great that the slightest pressure gave rise to great pain, preventing the detection of a tumor if such then existed. Rectal and vaginal examinations revealed nothing but tenderness high up posterior and to the right of the womb. No induration or fluctuation was felt. The abdominal muscles were more tense on the right than on the left side. There was decided impairment of the percussion resonance in the right iliac region. An area of absolute dullness (supposed to be hepatic) extended from the ribs to the crest of the ilium.

October 30, 11 A. M. The temperature was rather high on the 28th and 29th (see tabular view). The abdomen was distended and very tympanitic, obscuring the dullness detected at a previous visit. About that time she first began to lie with her right leg flexed. The tenderness was much less. Temperature of room, 70° . Axillary temperatures, right, 99.5° ; left, 99.8° ; pulse, 98; respiration, 28. Temperature of iliac regions, right, 100.2° ; left, 99.8° . Temperature of anterior fifth intercostal space, right, 98.2° ; left, 98.4° . She complained of almost daily chilly feelings, diarrhoea, and profuse perspiration. An examination of the urine failed to detect albumen or sugar.

November 3, 11 A. M. Temperature of room about 70° . Axillary temperature, 96.4° ; pulse, 88; respiration, 24. Temperature of iliac region, right, 95.9° ; left, 96.4° . She had a severe chill on the evening of the second inst. She perspired freely, vomited occasionally, ate but little, looked miserable, and was emaciating rapidly. The pulse was intermittent.

8th, 12 M. Temperature of room, 96.4° ; pulse, 120; respiration, 36. Temperature of lumbar regions, right, 96.8° ; left, 97.6° . There was decided induration, and a sense of fluctuation in the right lumbar and iliac regions. The great tympany somewhat obscured the fluctua-

tion. The bowels were opened twelve times during the night. The stools contained considerable pus.

9th. She died during the afternoon from exhaustion.

Tabular view of the temperature, pulse, and respiration, taken morning and evening, from October 27 to November 9, by Dr. Everett.

October 27, A. M. Temp. 100.5°; pulse, 104; resp. 27. P. M. Temp. 101.5°; pulse, 102; resp. 28.

28th, A. M. Temp. 100.8°; pulse, 96; resp. 24. P. M. Temp. 103.5°; pulse, 104; resp. 26.

29th, A. M. Temp. 102.5°; pulse, 108; resp. 32. P. M. Temp. 102.8°; pulse, 96; resp. 32.

30th, A. M. Temp. 102.2°; pulse, 90; resp. 24. Temp. P. M. 101.5°; pulse, 90; resp. 28.

31st, A. M. Temp. 101.4°; pulse, 92; resp. 26. P. M. Temp. 100.6°; pulse, 98; resp. 30.

November 1, A. M. Temp. 100.2°; pulse, 96; resp. 28. P. M. Temp. 99.9°; pulse, 104; resp. 28.

2d, A. M. Temp. 99.2°; pulse, 92; resp. 26. P. M. Temp. 98.8°; pulse, 80; resp. 28.

3d, A. M. Temp. 96.8; pulse, 88; resp. 24. P. M. Temp. 97.4°; pulse, 114; resp. 32.

4th, A. M. Temp. 97.4°; pulse, 102; resp. 32. P. M. Temp. 103.5°; pulse, 112; resp. 36.

5th, A. M. Temp. 99.8°; pulse, 98; resp. 28. P. M. Temp. 101.8°; pulse, 104; resp. 32.

6th, A. M. Temp. 98.8°; pulse, 88; resp. 28. P. M. Temp. 100.8°; pulse, 116; resp. 32.

7th, A. M. Temp. 101.4°; pulse, 84; resp. 40. P. M. Temp. 101.8°; pulse, 92; resp. 38.

8th, A. M. Temp. 100.8°; pulse, 122; resp. 40. P. M. Temp. 101.2°; pulse, 106; resp. 42.

9th, A. M. Temp. 99.2°; pulse, 108; resp. 40.

Post-mortem examination was made twenty-four hours after death by Dr. Everett and myself in the presence of Drs. Hickman and Moylan. The brain was not examined. Great emaciation, the chest and abdominal walls containing only traces of fat.

Thorax.—Numerous old and recent pleuritic adhesions were found, being more abundant on the left side near the heart. Several ounces of serous fluid were in each pleural sac. There were some adhesions between the left pleura and pericardium. Each lung contained several small abscesses filled with pus, and the bronchi were filled with pus and

frothy mucus. There were no evidences of pericarditis, except over a small area of the left ventricle, where a recent deposition of lymph was found. Only a few drachms of serous fluid were in the pericardium. The right side of the heart contained a large chicken-fat clot. The right auricle and ventricle were normal, and the valves healthy in appearance. The left auricle and ventricle were dilated and hypertrophied, and the latter contained a mixed fibro-chicken-fat clot, which did not extend into the aorta. The appendix of the left auricle was greatly hypertrophied, and its anterior tip lay in contact with the chest-wall. Its systole gave rise to the presystolic pulsation seen in the left second intercostal space. The mitral valves were very much diseased by ossific deposit, which constricted the orifice and rendered the valves incompetent. The auricular surfaces of the valvules were very rough. One of the valves was contracted and almost completely transformed into bone, but the other, being of a leathery thickness, vibrated with the current of blood. The valves at the aortic orifice were competent but thickened, and presented a leathery appearance. Adhesions had taken place between their free margins near their attached borders and constricted the orifice.

Abdomen.—The omentum contained but little fat. The peritoneum presented no evidence of general inflammation. In the right lumbar and iliac regions general adhesions surrounded a fluctuating tumor. The upper, upper anterior, and lateral boundaries of the adhesions were formed by the right lobe of the liver. An abscess sac containing about six ounces of pus and sloughing tissue was found immediately surrounding the appendix vermiformis, which had sloughed off, leaving an opening into the cæcum large enough to insert a lead pencil. Another opening into the cæcum, about an eighth of an inch in diameter, was discovered near the attachment of its appendix. No foreign body or piece of concrete fecal matter was seen. The cæcum was almost black, and partially gangrenous. The ascending colon contained several ounces of pus. Pus was seeking another outlet along the course of the psoas muscle, having found its way as far as the right inguinal region. The abdominal wall in the right lumbar region was rapidly breaking down before the suppurative process. The crest and spine of the right ilium were also laid bare by the dissection of burrowing matter. The adhesions included the right lobe of the liver, abdominal wall above the crest of the right ilium, cæcum, lower portion of the small bowel, lower part of the ascending colon, and the right kidney. The right lobe of the liver was very much enlarged, extended

downward to within one inch of the crest of the ilium, and with the abscess connected with the appendix of the cæcum, completely filled up the right lumbar region. The entire liver weighed eighty ounces. Its right lower margin was adherent to the abdominal wall over a space of about two inches in diameter. The adjacent portion of the liver contained numerous small abscesses all communicating with one large one, whose contents had begun to press upon the abdominal wall in the right lumbar region. Several ounces of pus had collected in this situation, and accounted for the fluctuation that had been detected a few days before death. Before the hepatic abscess was opened with the knife, when the liver was raised, pus was seen running from it into the sac or abscess surrounding the cæcum. The spleen was congested and the seat of two or three small abscesses. The kidneys, pancreas, and the womb and its appendages were nearly normal in appearance.

Remarks.—Dr. Broadbent, in discussing an article by Balfour on “Hæmic Murmurs,” said “he did not believe that, as a matter of fact, the auricular appendix” (left) “ever lay beneath the chest-wall at the point in question” (left second interspace). “He had examined every case of mitral stenosis he had seen since the publication of Dr. Balfour’s book” (in which auricular pulsation is described), “and had never been able to satisfy himself of its presence. He could not, indeed, understand how pulsation of the auricle could be felt through the chest-wall.” Pulsation in the left second intercostal space he attributed to the pulmonary artery.¹

In the case of the woman, an account of whose clinical history and pathological lesions is given in this paper, a murmur was heard in the pulmonary artery differing in character from those heard over other portions of the chest. Apparent presystolic pulsations were seen also in the left second intercostal space. At the autopsy the auricular appendix was found winding around a normal-sized pulmonary artery and lying against the chest-wall in the left second intercostal space. Dr. Balfour believes that so-called hæmic murmurs heard over the pulmonary artery are due to mitral regurgitation, the left auricle and ventricle being dilated, and the appendix of the former lying in contact with the chest-wall, and thus acting as a convenient medium for conducting the murmur to the ear at this point.² His explanation of the production of the murmur holds good in the present case.

¹ British Medical Journal, August 26, 1882.

² Ibid.

The older writers on the diseases of the heart stated that the mitral presystolic murmur was very infrequent and difficult of recognition. Instead of agreeing with these statements, I am inclined to favor the more recent views expressed and urged by Dr. Austin Flint, Sr., that this murmur is of comparatively frequent occurrence, and is usually easy of recognition. I do not wish to be understood as meaning that it is as common as the aortic, or mitral regurgitant, murmurs. Flint says he has met with many hundreds of examples of this murmur in hospital and private practice.¹ Two varieties of the mitral presystolic murmur exist, the rough or vibratory, and the soft, the former being far the more frequent, and easily mistaken by an inexperienced or careless observer for mitral regurgitation. An excellent *résumé* of the whole subject by Dr. Flint, of N. Y., may be found in the April number of the "American Journal of the Medical Sciences" for 1882. Attention to one point, when the rough mitral presystolic murmur exists with the mitral regurgitant, has materially aided me in the differential diagnosis. In all these cases the beginning murmur (presystolic), when listened to over the left fourth costal cartilage, is harsher than the one (mitral systolic) which immediately follows it, and when the chest is auscultated in the axillary region, and posteriorly at the lower angle of the left scapula, only the softer mitral regurgitant murmur is heard. In rare instances I have been able to hear the rough mitral presystolic murmur in the anterior portion of the axillary region, but never over the posterior surface of the chest.

The diagnosis of hepatic abscess associated with some peri-uterine suppuration was ventured during life, but as she had been sick several weeks before coming to the hospital, the duration of the hepatic enlargement could not be ascertained. The clinical facts in favor of abscess of the liver were the suddenly developed jaundice, obstinate constipation at first, followed by alternating diarrhœa and constipation, pain in the right side, and the extension of liver dulness from the ribs to the crest of the ilium. The evidences in favor of suppuration around the womb were very strong. These were, the sudden suppression of the menstrual flow so soon after subjecting the lower portion of the body to the prolonged cooling effects of marble, and this in turn so soon followed by intense and agonizing abdominal pains, which shortly after became localized in the right iliac and lumbar regions, and the great tenderness in the right iliac fossa associated with the

¹ American Journal of the Medical Sciences for April, 1882, p. 444.

pinched features of peritoneal inflammation. It was certain that suppuration was going on in some portion of the abdominal cavity. Rectal and vaginal examinations failed to detect pus in the pelvis, and, on account of the great tympany, and the extreme sensibility in the right lower portion of the abdomen, fluctuation in the right lumbar region was not detected until a few days before her death.

In the light of the pathological lesions seen at the autopsy, is it possible to determine the seat of the primary suppuration? When the liver was raised by the hand, a communication was seen between the hepatic abscess and the sac formed around the cæcum, and pus escaping from the former to the latter situation, yet it should be borne in mind that the thin wall of the liver abscess was easily ruptured, and might have occurred during the examination, notwithstanding that great care was taken to prevent such an accident. The appendix of the cæcum had sloughed, and was nearly or completely detached, it being in a condition similar to that in which it is found in perityphlitis. On the other hand, the second opening in the cæcum near the attachment of its appendix, the large size of the abscesses of the liver, and their connection with all the smaller ones, the hepatic suppuration being limited to the right of the right lobe, the adhesions of the liver to the abdominal wall, and the formation of a sinus beginning at this point, which was above the contents of the sac surrounding the cæcum, and extending to the right inguinal region, by the pus burrowing from the liver, when taken in connection with the former history of malarial fever (intermittent), the onset of her fatal illness being preceded by exposure to cold, the early jaundice, and the fever and stomachic derangement, primarily having been only of a few days' duration, point, to my mind, almost conclusively, to the suppuration having been of hepatic origin. I have been unable to find a recorded instance of circumscribed abscess of the liver following perityphlitis. Abscess of the liver not infrequently opens into the bowel.

Would an operation after the patient's admission into the hospital have been justified? During her stay there the seat of suppuration was too ill defined, in view of her anæmic condition and severe cardiac complication, to hazard any surgical procedure.

The *surface temperature* of the abdomen was usually above that of the axilla, and exceeded the chest temperature one to two degrees. It indicated inflammation in the abdominal cavity, but strange as it may seem, the temperature of the healthy side equalled, and for a few

days preceding death, exceeded that of the diseased. No surface temperatures were taken on the patient until suppuration was undoubted.

November 23d, 1882.

19. *Small tumor of the head of the pancreas.*

Presented by Dr. E. T. BRUEN.

The mass here exhibited occupies the head of the pancreas, and is about as large as a pullet's egg, or about two-and-a-half inches in diameter. When removed from the cadaver, it was closely adherent to the common bile-duct. The growth is of a colloid nature, but its histological character is as yet unsettled by microscopic examination. There were no secondary deposits in any other viscera, as is the rule in colloid growths. The patient was a male, æt. about 65 years. For two years prior to date of his death, there had been intermittent attacks of jaundice. This had been attributed to catarrh of the biliary duct, which had developed itself during an attack of malaria. The case was under my care for two months. At first the symptoms of chills and fever led to treatment for malaria; but as the jaundice gradually deepened, treatment for the supposed catarrhal state of the bile-duct was instituted. The futility of a well-directed and usually successful treatment was the special indication of some more substantial cause. Hepatic trouble or biliary calculi were readily excluded in this case, but there was nothing to fasten suspicion on the pancreas. There was constipation, with clay-colored stools. There was a febrile movement of the hectic type—the so-called hepatic fever, accompanied by irregular chills. The intestinal indigestion, so valuable a symptom in many cases of pancreatic disease, was not marked.

Indeed, without exception, the negative results of the clinical record were of the most discouraging character. The case is presented because each case of pancreatic disease ought to be recorded, so that many cases viewed collectively may contribute to the clearness of a future distinct picture of pancreatic disease.

Dr. Formad said that he had now on record the notes of five or six cases of cancer of the pancreas with marked jaundice, where he had made the autopsy. It seemed to him that the jaundice was most persistent in primary pancreatic carcinoma from pressure on the bile-duct. He was present at the autopsy of this case, and did not think that the tumor was carcinomatous, as there would then probably have been

secondary growths in the liver. The growth was probably a cystic colloid.

Dr. Bruen would like to call the attention of the Society to the fact that he had about a month ago presented a specimen of carcinoma of the pancreas, and had then referred at length to some forty cases of jaundice due to primary carcinoma of the head of the pancreas, lately reported by another observer. From these records it was demonstrable that jaundice was an invariable symptom of primary hard carcinoma of the head of the pancreas, while it was uncommon when the disease was secondary or affected other parts of the organ. *January 11th, 1883.*

20. *Diaphragmatic hernia.*

Presented by Dr. Tyson.

The patient from whom these specimens were removed was a German between 27 and 28 years of age at his death. He first consulted me June 21, 1880. He had then been ailing since February, 1878, and had been unable to work in that time. At the date referred to, he was stretching, when his wife suddenly pretended to tickle him. He quickly threw down his arms, and at that moment felt a sensation of pain on the left side in the neighborhood of the heart. At the same time he felt faint and cold. In five or ten minutes these sensations passed away, but he remained very much frightened. When he first visited me he complained of shortness of breath and beating of the heart, although the latter was better when he was quiet. He could not even walk across the floor without becoming completely out of breath, but he said he was not short of breath when the accident first occurred. He had also a peculiar puffing expiration which did not occur, however, with every act of expiration, but once in four or five. This was so characteristic that one of my friends, to whom I sent him, spoke of him as my "puffing German." There was no cough.

Physical examination revealed, on inspection, almost total absence of movement of the chest-wall in the upper half, the respiration being almost purely abdominal. The upper percussion border of the heart corresponded with the junction of the third rib with the sternum, and the right border with the middle of the sternum. The apex beat was in its normal position, but was more diffuse than in a strictly normal state. Pulmonary percussion appeared normal except below the left scapula, where resonance was less than in the corresponding situation on the right side. *Vocal fremitus* was, however, impaired over the

whole of the left lung. There were no abnormal cardiac murmurs. His pulse was 72 after the examination was over. He was treated at various times with digitalis, bromide of potassium, chloral, tonics, etc., and even a blister was put over his heart, with the view that probably there was some cardiac or pericardial affection, although the physical signs were wanting. There was no improvement, although at times the dyspnœa, which was the most distressing symptom, seemed sometimes less; but it always interfered with any exercise whatever. He would walk sometimes half a mile with great difficulty; notwithstanding this he was encouraged to take exercise. His sleep was unsatisfactory; he would dream, and wake up in great fright. He could always lie upon his left side with more comfort than upon his right.

He continued under my observation for a year. I saw him in the latter part of August, 1881, when he seemed a little better. After that I heard nothing of him until I learned of his death, which occurred December 15, 1882, from intestinal obstruction.

At the *autopsy* it was ascertained that about twenty inches of the large intestine, with its corresponding mesentery and almost the whole omentum, had ascended through the œsophageal opening of the diaphragm into the left pleural sac, encroaching upon the space occupied by the left lung until the latter was compressed into the apex of the left pleural sac and was reduced to a cylindroid mass about fifteen centimetres in length and half as many in diameter. There was no hernial sac. The heart was displaced to the right, but was otherwise normal; the liver was slightly fatty, but the other viscera were natural.

It is not unlikely, at the moment referred to in the history, when the patient threw down his arms, that a small portion of omentum or mesentery slipped through the œsophageal opening, and that subsequently and more or less gradually the vacuum tendency of the pleural sac in each act of respiration caused the remainder of the mass to be drawn in until the entire cavity was occupied. This accounts for the fact that there was no dyspnœa at the beginning, but that it gradually increased as the thoracic space was intruded upon.

In vol. 81, 1882, of "Virchow's Archiv" will be found an exhaustive article on Diaphragmatic Hernia, in which 291 cases are collated. In a somewhat hasty examination of this paper I have been unable to discover a single case so long under observation as this—nearly four years. Many cases were discovered at the autopsy and were unsuspected, others were congenital, and others were traumatic.

January 11th, 1883.

21. *Ulcer of the stomach in the anterior wall; adhesions to the abdominal wall; hemorrhages and pain; death from the morphia habit.*

Presented by J. H. MUSSER, M.D.

I am indebted to Dr. J. Henry Musser, of Lancaster County, for the privilege of presenting this specimen. I abstract from his notes the following history: I. J., aged 71 years, millwright. Fourteen years before his death he was attended by the doctor for hæmatemesis occurring after three days of nausea. Over a pint of blood was vomited, and considerable was discharged by stool the next day. In ten days he returned to work and enjoyed good health for two years, with the exception of occasional attacks of indigestion. Then a recurrence of nausea, followed by a profuse discharge of blood, per anum, occurred. He made a full recovery, but pain after eating was now noticed. Two years subsequently he bled very much from a wound of the foot, and was much prostrated thereby. This was succeeded by severe pain after eating, with marked epigastric tenderness. The constant use of narcotics alone gave him relief. November, 1873, he was again under Dr. J. Henry Musser's care, and was treated for gastric ulcer without any avail, save when he exhibited narcotics. He resorted to the use of morphia, and continued its use during the remainder of his life. Change of diet did not influence the pain, and he partook of laborers' fare. In 1877, a small tumor to the left of and several inches above the umbilicus, hard, tender, and apparently involving the abdominal wall, was discovered. For several years he worked in a country saw-mill, using one drachm of morphia a month. Heavy lifting aggravated his pain. August, 1882, the doctor was again called in, to find the patient confined to bed, much emaciated, and with complete anorexia. Death took place three weeks later, during which time he had taken no nourishment, but had used morphia freely. The habit had been acquired, and his friends thought he suffered but little pain. The only anatomical alteration noted at the autopsy was the change in the stomach. The organ was enlarged and contracted (hour-glass) towards its fundus. The anterior wall, one-third of the distance from the fundus, and at the point of constriction, was adherent to the abdominal parietes to the left of the median line, three inches above the umbilicus. Opposite to the point of adhesion in the mucous membrane there was a small ulcer two lines deep and

six in diameter, with clean-cut edges, a healthy floor, and surrounded by cicatricial tissue for a radius of one inch. Undoubtedly the ulcer would have healed entirely, had not the morphia habit cut off the patient.

Remarks by Dr. J. H. Musser. It is plausible to theorize that the first hemorrhage, as well as the profuse foot hemorrhage, was due to the patient having been of an hemorrhagic diathesis, and that the second gastric hemorrhage was from the same cause, and was attended by a hemorrhagic infarct, with subsequent development of the ulcer. Especially is this possible, as it was only after the second hemorrhage that pain occurred, and this symptom is never absent, "except in cases which run a rapid course." [Da Costa.] *January 11th, 1883.*

22. *Five cases of carcinoma.*¹

Presented by Dr. GUY HINSDALE.

CASE I. Carcinoma of the stomach and pancreas with metastases in the liver and lungs.

Carl W., æt. 44, a moulder, and a German, was admitted to the hospital October 23, 1882. He had never been ill before, and his symptoms had developed during the two weeks previous to his admission. The only history obtained was that his bowels had been regularly opened and that he had not had any vomiting, although he had suffered from a great deal of pain in his stomach and at the lower margin of the left ribs.

Cachexia was marked. He had a haggard look. He also coughed and had a slight expectoration. Friction sounds could be heard over the base of the left chest posteriorly. The abdomen was soft; a tumor was distinctly felt in the left hypochondriac region; the spleen was enlarged and distinctly felt.

The patient lived twenty-six days after admission—just six weeks from the first intimation he had had of his disease.

Notes taken ten days before death state that the tumor in the left hypochondriac region is becoming more distinct, is smooth in outline and apparently descending. Two inches above the umbilicus a distinct hardness is felt, its area being continuous with the area of liver

¹ This paper was submitted in competition for the prize open to resident physicians of hospitals, and received the award.

dulness. These tumors are sore to the touch and the seat of great pain at all times. He had no desire for food, and a few oranges was all that he cared to eat.

During his entire illness the patient did not vomit and his bowels were regular. He grew more haggard and suffered more and more pain. Chloral and bromide only aggravated it, and morphia had to be given in large doses. His sufferings were so great that he would gladly have committed suicide had he been able. Death occurred at night after a severe attack of gastric pain.

At the autopsy, fourteen hours after death, the body was found greatly emaciated; the skin having a dark-yellowish hue; a slight effusion was found in both the pleural and abdominal cavities. The latter fluid was reddish in color. The pericardium also contained fluid. Upon the right side there were slight pleuritic adhesions; none on the left, but the walls of the fissure between the lobes were adherent. The roots of the lungs were the seat of carcinomatous nodules; the base of the left lung adjacent to the diaphragm and the pleura beneath the ninth and tenth ribs were also the seats of carcinomatous deposits. The thyroid gland was hypertrophied, but not carcinomatous.

The cardiac extremity of the stomach was the seat of an extensive carcinoma; the cardiac orifice was not involved and the mucous membrane not destroyed. The pyloric end was intact.

The pancreas was also the seat of carcinomatous growth. The liver was of a dark, mottled color and showed numerous metastatic growths in an early stage of development, which were distributed through the interior of the organ as well as on its periphery. The liver weighed five pounds nine ounces. The spleen was greatly enlarged and the seat of small secondary deposits. The organ was shown in connection with the stomach and pancreas, from which it has not been separated. The kidneys were normal.

Microscopic sections of the secondary growths in the liver and thyroid gland were presented at the meeting.

CASE II. Scirrhus carcinoma of the stomach. Metastases in the liver.

James S., aged forty, entered the hospital September 28, 1882, with the following history: Since the beginning of his illness, two months previous to admission, he had lost forty pounds of flesh, and the color of his skin had been steadily growing sallow. From the start, his chief symptom had been vomiting, which took place frequently before

breakfast, when a clear, watery fluid was expelled. Sometimes, however, the vomiting occurred from one to two hours after meals.

Upon examining the abdomen a tumor was distinctly felt upon the left side, above the umbilicus and just below the margin of the ribs. The tumor moved with respiration and was painful upon pressure. An examination of the remainder of the abdomen revealed nothing abnormal. Albumen was present in the urine.

Inquiry into his family history revealed the fact that his mother had died of carcinoma of the stomach.

After admission the tumor increased in size until it extended to the right of the median line, and downwards to within an inch of the umbilicus. The patient became steadily weaker, vomiting occasionally and passing clay-colored stools, until the thirty-eighth day after admission, when he died.

At the autopsy the heart, lungs, kidneys, and spleen were found in a normal condition; the stomach was very much dilated and contained undigested food, its pyloric extremity being the seat of carcinoma. The pylorus was very much thickened and admitted the thumb with difficulty. The gastric walls at the seat of the carcinoma had been destroyed to such an extent that the cavity of the stomach was separated from the abdominal cavity only by a delicate membrane which was so easily torn that a portion of the contents of the stomach escaped before that organ was removed.

The liver was then detached, and its under surface displayed four large bosses of secondary carcinoma. The liver weighed three pounds fourteen ounces. The gall-duct was unobstructed.

In this case the stomach was undoubtedly the primary seat of the disease. It is proper to add that the liver not being enlarged and the carcinomatous deposits being upon its under surface, this secondary carcinoma was not suspected during life.

CASE III. Carcinoma of the stomach. Gastro-colic fistula.

Christopher W., a German laborer, æt. 66, was admitted to the male medical wards of the Episcopal Hospital on June 13, 1882, and was a patient of Dr. Morris J. Lewis. The patient's health had been good, until four months previously, when he vomited some dark bloody matter. About this time he also experienced pain in the epigastrium, which was not constant, and which was sometimes dull, sometimes severe, as though stabbed with a knife. The pain occurred after eating, and especially after breakfast. He had vomited only once since the first

attack, when the ejecta were white. Jaundice began three weeks before admission; the skin was dry, and the body not much emaciated. The tongue was slightly coated, the bowels were opened regularly, and the stools clay-colored.

Upon examining the abdomen, the liver was found to be slightly enlarged, and what was apparently its left lobe could be felt in the epigastrium. Three inches to the left of the umbilicus, and four inches above it, a large nodulated mass three inches in diameter was felt, freely movable, and slightly painful on pressure. To the right of the umbilicus and slightly below it, a second mass was felt, much less resistant than the former, and of smaller size. No dulness was found in the splenic region. The inguinal glands were slightly enlarged; the axillary glands remained normal.

There was no cough. A faint, low, systolic murmur could be heard at the aortic cartilages. The urine was dark yellow; sp. gr. 1.010; alkaline; no albumen.

During the patient's first month in the hospital, vomiting, which occurred at first every five or six days, finally took place at every meal, the ejecta consisting simply of the food which was taken. The pains grew more persistent and severe, emaciation progressed rapidly, and jaundice increased. The patient became steadily weaker, and fell once or twice on attempting to walk alone to the bath-room.

During the last month of his illness the tumors increased in size, and the jaundice became more marked. The patient frequently at once vomited everything he took; he felt miserable, and his mind wandered. August 12 the patient died easily, and without giving much warning, respiration failing first, when the heart-beats, which had become quite irregular, finally ceased.

At the autopsy, twelve hours after death, the body was found greatly emaciated, and the skin and all internal organs markedly jaundiced. Lungs normal, except for old adhesions at the right apex. Heart normal. Upon opening the abdominal cavity, the stomach was found with its long axis perpendicular, the pyloric extremity being at the left of the median line, about two inches below the lower border of the ribs. A carcinomatous mass, measuring 2 x 3 inches, was situated at the pylorus.

The transverse colon was infiltrated with the new growth, and firmly adherent to the pylorus. Upon removing the tumor, together with the adherent structures, the following facts were noted:—

It was noticed that upon allowing water to flow through the portion

of colon exsected, it escaped through the pylorus, and *vice versa*. The calibre of the pylorus was not markedly contracted, although rendered rigid by the new growth which surrounded this part, except at the shorter curvature. The colon was contracted very much, barely admitting the index finger. Upon laying the colon and stomach open, the new growth was seen to encircle the pylorus, except for about one-fifth of its upper part, as above mentioned; there was marked ulceration of the mucous membrane at this part, and a communication with the colon was discovered; the opening was ragged, and was of sufficient size to admit the little finger. Marked ulceration of the upper wall of the colon and ulceration of the mucous membrane were found. There was no dilatation or hypertrophy of the walls of the stomach. The liver was large, friable, and very dark in color. The gall-bladder was enormously distended with about three ounces of dark-green bile. A large mass of carcinomatous glands occupied the centre of the abdominal cavity, immediately over the bodies of the vertebræ, the new growth infiltrating the mucous membrane and walls of the duodenum, surrounding and obliterating the common bile-duct, and that of the pancreas. The kidneys presented nothing abnormal, other than that the left contained a cyst of the size of a filbert.

An enlarged gland, about the size of a small egg, and moderately movable, was situated upon the right of the median line below the level of the umbilicus. The mesenteric glands were slightly enlarged; the spleen was small, and its hilus the seat of a hard, calcareous plate. The organ was displaced, lying close against the diaphragm, some distance from the chest-wall, allowing the lung to extend over and below it for about two inches, thus accounting for the perfect resonance in the splenic region.

CASE IV. Carcinoma of the rectum. Colotomy.

John M., a stonecutter, æt. 71, was admitted to the Episcopal Hospital October 26, 1882. He stated that he had always been healthy and strong until three months previously, when the movements of his bowels began to be irregular. At first, three or four days would pass without a passage, although he experienced the desire for stool. Then diarrhœa followed, six or seven passages occurring daily, and often without the patient's control.

Upon admission, the belly was found soft, the liver and spleen not enlarged, and the lungs clear. A soft systolic murmur was heard over the aortic valves. The urine contained albumen and hyaline casts,

was acid, and its sp. gr. 1018. The patient also had right hydrocele. He denied syphilis and was apparently without specific history.

Exploration of the rectum revealed a malignant stricture one and a half inch from the anus, which firmly resisted the full entrance of the finger.

On account of the patient's advanced age and the extent of the disease, operative measures were deemed inadvisable. The passages, however, became more difficult, the contents of the intestines accumulated, and hiccup and stercoraceous vomiting demanded relief. This was afforded by colotomy, which was performed by Dr. John H. Packard. An oblique incision was made on the left side, its middle point being two and a half inches above the highest point of the crest of the ilium and directed toward the middle of Poupart's ligament. The bowel was easily reached, secured by a curved needle and brought to the surface, where a longitudinal incision was made in the gut and the edges stitched to the skin. An enormous amount of semi-solid feces escaped through the opening; the abdominal distension and the hiccup and vomiting subsided. Death ensued, however, twenty-four hours later.

At the autopsy the wound was found in good condition and the stitches holding.

Upon opening the abdomen the descending colon, enormously distended, was the most prominent object. The incision was found to have been made longitudinally about three inches from the point where the colon bends downwards. Feces readily passed from the opening in the side upon the slightest pressure on the distended colon. Exploration per anum showed the stricture to be impermeable. The rectum and bladder, including the entire carcinomatous growth, were then removed. This was accomplished with the greatest difficulty, since the carcinoma nearly filled the cavity of the true pelvis. The posterior wall of the bladder was found to be densely infiltrated, and the stricture, which was situated about one and a half inch from the anus, admitted even a pencil with difficulty. All the other viscera were found in good condition and no peritonitis had taken place.

Microscopic sections show the growth to be scirrhus carcinoma.

CASE V. Carcinoma of the rectum. Secondary carcinoma of the liver.

Christian H., also a German, was admitted to the hospital July 6, 1882. He was emaciated, weak, and cachectic. He stated that for

twenty-seven years he had been perfectly well ; that two months previously he noticed that he had been losing flesh, and at the same time discovered a growth at the anus which was small, but which gave him a great deal of trouble at stool. Two weeks later he noticed that his feet and legs became swollen. It was not until his admission to the hospital that he became aware that there was anything wrong with his abdomen.

The patient stated that his father died at the age of forty-nine of a disease of the stomach of three or four years' standing ; that his skin was yellowish and his body very much emaciated when he died, and that "his stomach was stopped up on one side."

The anal growth, for which primarily he sought treatment, was excised, and on examination proved to be an adenoid type of malignant disease. After admission the patient vomited occasionally, but the ejecta consisted merely of the food which he had taken five or ten minutes before. The bowels were opened twice daily ; the passages were usually solid and the movements accompanied by pain. Dyspnœa was easily brought on by any exertion. The heart sounds at that time were normal and the beats 112 per minute. The patient gradually lost flesh and strength. His skin became more sallow but not jaundiced, his death occurring August 14th.

The following notes were taken during the last week of his life : The œdema has extended gradually to the ribs, which bulge considerably upon the right side. The liver is of enormous size, producing a marked prominence of the epigastrium, nodular and comparatively painless excepting upon pressure, and rising and falling with the diaphragm ; its area of dulness begins anteriorly at the right fifth rib and extends to a point one inch below the umbilicus. There is ascites. The spleen, lungs, and heart are apparently normal. During the four days preceding his death, however, a soft, double murmur was heard at the apex. The patient's heart became intermittent, dyspnœa and hiccough came on, and he gradually sank.

At the autopsy, twelve hours after death, it was noted that there were emaciation, double inguinal hernia, and a deformity on the left side, from a fracture of the tenth costal cartilage, which had occurred when he was twenty years of age ; old and strong pleural adhesions of the right lung ; no nodules present ; no pleural effusion. Pericardium normal, and no effusion. Heart contained two chicken-fat clots in both ventricles, the right slightly dilated ; valves normal. Stomach, spleen, and kidneys normal.

The liver was enormously enlarged, filling the whole upper portion of the abdominal cavity. It was slightly adherent to the diaphragm and adjacent viscera in front, but was more closely adherent posteriorly. It was studded with carcinomatous nodules, varying in size from that of a filbert to that of an egg, often coalescing, and pretty equally distributed over the surface. The whitish color of these nodules was in strong contrast with the dark color of the liver, giving it a variegated appearance. The right lobe was six inches in thickness, and almost globular. The liver weighed ten pounds four and a half ounces. The gall-bladder was normal, and its duct patulous; the mesenteric glands were slightly involved.

The walls of the rectum, six inches from the anus, were infiltrated with the new growth, but did not present any point of ulceration.

In this case the carcinoma originated in the rectum, and by means of the portal circulation and lymphatics, metastases occurred in the liver. The secondary growth was very rapid, almost entirely painless in itself, and disturbing the patient only by mechanical irritation of the stomach, and interference with the circulation. Jaundice did not occur.

Microscopic sections were made, first, of the portion of the growth excised at the anus; second, of the mass found six inches from the anus; and, third, of the carcinomatous nodules in the liver.

The first specimen is an example of the early involvement of the part. It shows an inter-cellular substance of fibrous tissue having cavities or follicles lined with cylindrical cells. It has the appearance of ingrowing follicles of the rectum. It is an example of adenoid growth, and resembles an aggregation of Lieberkuhn's crypts, a resemblance which is so marked in some cases as to make it almost impossible to distinguish it from natural tissue. These cases of malignant adenoid disease are characterized by an exceedingly rapid growth, quite in contrast with the slow progress of papillomata and villous tumors, which closely resemble them in structure.

The second specimen was taken from the mass one inch by one and a half inch in size, found high up in the rectum. The section shows the encephaloid form of the disease, and is simply a further stage of the same growth.

The third specimen is a nodule from the liver. It resembles very much the second. It shows a stroma of fibrous tissue with alveoli, lined by epithelial cells, the peripheral cells retaining their columnar shape. The reproduction in the liver of the same follicles found in

the primary disease of the rectum has been frequently noted by Moxon and others.¹

Rectal cancer starts, I take it, as an hypertrophy of the follicles of the rectum, involving subsequently the deeper layers of the bowel. Its surface is marked by a regular cylindrical arrangement of the epithelial cells, suggesting at once adenoid growth. The structure of the central portion is less suggestive of glandular tissue, but lacks enough connective tissue to designate it as scirrhus.

According to Cripps,² the morbid action commences as an infiltration of the submucous tissue, the follicles in this portion of the mucous membrane being three or four times their usual length, the lining epithelium large, the boundary line between the cells being clearly defined. The subjacent retiform tissue is enormously thickened, and the seat of the bulk of the new adenoid growth. Subsequently, the delicate layer of mucous membrane becomes ulcerated, and we have the more advanced fungoid form of the disease.

An interesting study of the metastasis of tumors, examples of which I present, is given in the graduating experimental thesis of Dr. Henry Wile, of the University of Pennsylvania, portions of which were printed in the *Phila. Medical Times* during the summer of 1882.

We cannot hold with Billroth that the secondary deposit is due to a common cause, viz., the dyscrasia of the blood, and without any anatomical dependence. We must hold the secondary tumors to be the result of the development of tumor emboli carried through the lymphatics or the bloodvessels to points from which they develop with a central independent growth.

In the cases presented, as in almost all cases, the etiology of the primary growth is obscure. To be sure, in chimney-sweepers' cancer of the scrotum, the origin is plainly and confessedly a local inflammatory one; but, as Prof. Gross said in 1839, "very frequently, it is true, the disease arises imperceptibly, without local injury or obvious constitutional derangement; but this certainly does not prove that inflammation is not concerned in its production." *March 8th, 1883.*

¹ Bryant's Surgery, Phila., 1879, p. 114.

² Cancer of the Rectum, p. 63.

23. Specimen of invagination of the ileum.

Presented by Dr. C. M. WILSON.

The specimen was taken from a patient who died in the Jefferson College hospital two weeks ago. Her symptoms before death made an absolute diagnosis difficult. She came in with the history of an attempted abortion. She had had a severe attack of peritonitis one year previous to her present sickness, which had undoubtedly produced fixation of a portion of the ileum by binding it to the subjacent abdominal parietes. It is supposed that, in some attack of abdominal tenesmus occurring in her present sickness, a portion of the gut above was forced down into the fixed portion of the ileum, became strangulated, and eventually sloughed as you see. The intussuscepted portion is about eighteen inches long; by this sloughing nature has made efforts to throw off the invaginated portion and restore the continuity of the alimentary canal. The perforation you see at the point of constriction at the commencement of the intussusception is a gangrenous ulcer. The whole of the intestine included in the intussusception, and below the point of constriction is gangrenous. *April 12th, 1883.*

24. Carcinoma of the stomach and colon with cardiac lesions.

Presented by Dr. J. T. ESKRIDGE.

Peter Lawler, aged 60 years, Irishman, by occupation a dyer, was said to have enjoyed fair health until two years ago, when he suffered from a severe attack of inflammatory rheumatism. He knew but little of his family history, and could not give the cause of his parents' death. He never complained of heart disease. During the latter part of the year 1881, about eighteen months before his death, he first began to complain of pain in the epigastric region, attended by eructation of a sour, slimy liquid. Soon he experienced a sense of nausea coming on an hour or more after eating. About three months after the first appearance of symptoms of gastric disease, he began to vomit. At first vomiting occurred occasionally, but soon it took place several times a week, and finally once or twice each day. He lost flesh rapidly. In the early part of March, 1883, he was admitted into the wards of St. Mary's Hospital, when he came under my observa-

tion for the first time. He was very weak, and greatly emaciated. His pulse, of the Corrigan type, was eighty per minute. When resting in the recumbent posture, his breathing was quiet. Temperature was usually one degree below normal. The radial and temporal arteries were rather hard, and the latter were tortuous. A diastolic murmur and a systolic murmur were heard at the aortic orifice. The impulse of the heart was not very strong, and the left ventricle did not appear to be greatly enlarged. The lungs were emphysematous, and an area of impaired resonance, amounting almost to dullness, was discovered on each side of the spinal column, opposite the spines of the scapulæ. An indurated mass, apparently about the size of a walnut, more or less movable, was felt in the epigastric region to the right of the median line, and about midway between the ribs and the umbilicus. The growth was not sensitive to rather rough manipulation, and he had not experienced any pain for a number of months. His bowels were sluggish, and it required active agents to evacuate them. No tumor besides the one connected with the stomach was felt or suspected in any other portion of the abdomen. He vomited almost daily. The liver and spleen were not enlarged. The urine was free from albumen.

By securing daily evacuations from the bowels, and giving him nutritious, easily assimilated food, the vomiting nearly ceased. He improved and left the hospital the latter part of March. Early in April he was admitted to the Jefferson Medical College Hospital, where he came under my care the first of May. At that time he was eating but little, the abdomen was considerably distended by gas, and his bowels required repeated large enemata, or enormous doses of purgatives, to secure their action. On the fourth of the present month, he experienced great pain in the right iliac region, just to the right of the median line of the abdomen, midway between the pubes and umbilicus. Over and around the latter painful spot, a circumscribed highly tympanitic and sensitive area, about the size of a man's doubled fist, was observed. Circumscribed peritonitis was diagnosed. Large doses of morphia, administered hypodermically, were required to relieve pain. The stomach became irritable, and the peritonitis more general. He died during the afternoon of the eighth of May.

Sectio cadaveris four hours after death by the pathologist of the hospital, Dr. Morris Longstreth.

Thorax.—About seven ounces of perfectly clear serum were found in the pericardium. No evidence of pericarditis existed. Left side

of heart firmly contracted; right side relaxed, and contained considerable fluid blood. Right side of the heart and its valves normal. No lesion found at the mitral orifice. Free borders of the leaflets of the aortic valve are thickened and slightly contracted, allowing regurgitation to take place at the aortic orifice. Aorta atheromatous, dilated, and decidedly roughened near the aortic orifice. Left ventricle slightly hypertrophied.

Lungs deeply pigmented, and generally emphysematous; both congested posteriorly. Surfaces of both apices covered with patches of fibroid thickening. Abundant evidences of diffuse peri-bronchitis chronica were present. Bronchial tubes of the lower lobes of both lungs much dilated. Bronchial glands at the root of the lungs very much enlarged.

Abdomen.—On opening the abdominal cavity, considerable very offensive gas escaped from the upper part. On the right of the median line of the abdomen, from the umbilicus downward, the abdominal wall anteriorly was adherent to the omentum and intestine over an area of about five inches in diameter. The lower third of the abdominal cavity was filled with a yellowish-white cloudy liquid. The intestines were bound together by numerous adhesions. The stomach, which I show you, is small, and its coats are thickened. The hypertrophy of the wall of the stomach is slight at the cardiac end, but, gradually increasing, becomes considerable at the pyloric. The wall of the pylorus and adjacent portions of the stomach and small bowel is about one half inch thick. At this point the mucous surface presents several fungous-looking outgrowths. The small bowel, with the exception of about half an inch of the upper portion of the duodenum, appears normal. In the colon, about six inches from the ileo-cæcal valve, is a stenosis, barely admitting the end of my little finger. The wall of the colon at the point of narrowing, which extends three or four inches of the length of the bowel, is greatly hypertrophied, measuring about one-third of an inch in thickness. The colon, from its beginning to the point of constriction, is dilated into a large pouch, measuring four and a half inches in diameter. The dilated portion of the bowel presented a dark gangrenous appearance, distended by gas, and was adherent to the anterior wall of the abdomen, just to the right of the median line. The remaining portion of the large bowel appeared to be healthy. No enlargement of the mesenteric glands was observed. Liver, spleen, and pancreas were small and firm, but free from malignant growths. Both kidneys were reduced in size, contained a few small

cysts. their cortical substance was lessened, and their capsules were abnormally adherent in places.

Remarks.—It is worthy of remark that although considerable thickening and induration existed at the pyloric end of the stomach, the orifice remained sufficiently patulous to allow the food to come in contact with the intestinal juices. Another point of interest is seen in the existence of so great an amount of narrowing in the calibre of the large bowel with no symptoms, except easily obviated constipation, until a short time before the man's death. It seems to me remarkable that a bowel so dilated above the point of a narrow constriction should be able to respond painlessly to purgatives.

May 24th, 1883.

25. *Carcinoma (scirrhus) of the rectum.*

Presented by Dr. J. H. MUSSER.

At the autopsy of the person from whom these specimens were removed, made thirty-six hours after death, by Dr. W. E. Hughes, we found rigor mortis well marked, the body greatly emaciated, the skin of a yellow-earth color, and on the right buttock near the gluteal fold, the ragged, grayish openings of several sinuses which, we subsequently proved, communicated with a sac behind the rectum. This sac opened into the rectum, which at that point was greatly dilated. At the bottom of the dilated pouch in the posterior wall, towards the anus and two and a half inches therefrom, was a hard mass. This mass was the size of a silver dollar, involved two-thirds of the wall, but did not cause occlusion of the gut. The surface was ulcerated. The bowel towards the cæcum was dilated, the mucous membrane congested, and the muscular coat hypertrophied. The remainder of the intestine was normal, and the glands of the mesentery were only slightly enlarged. The liver weighed four and one-half pounds, was very fatty, and in the left lobe a secondary mass larger than a walnut was found. The apex of the left lung was the seat of a small area of catarrhal pneumonia; the base was bound down by rather recent adhesions. Further than this, the tissues presented no other changes than those due to wasting disease.

Microscopical examination revealed the two nodules to be of the nature of a hard carcinoma.

The patient, a female, 37 years old, had always been a dyspeptic and of a constipated habit. Her mother and one sister had died of carcinoma of the stomach, her maternal uncle of the same disease of the liver. I attended Mrs. — from October 11, 1881, to February 7, 1882. She died in May of the latter year. From the 5th of August, previous to my first visit, she had been suffering from so-called dysentery—twenty to thirty bloody and mucous stools with tormina and tenesmus. She had grown very feeble and lost much flesh. My notes state: "Abdomen flat, tender all over, especially in the fossæ, but no tumor noticeable." Dysenteric diarrhœa continued, and I may say these discharges kept up during my attendance; at times better, again worse. The mucus was always present; the blood, at times none, again slight or again in large amounts. The appetite was poor and her dyspepsia bad. The debility and emaciation progressed and the appetite became less. In November I detected a hard tumor, with apparently a raw surface, two inches and a half from the anus. December 19th I noticed the catarrhal pneumonia; December 31st the acute pleurisy. During December and January she complained of pains in the legs and of severe cramps at night. In January she began to complain of pain in the right hip posteriorly. Local and internal remedies scarcely relieved it, and finally the post-rectal abscess discharged. Death from exhaustion ended this career of horrible suffering. I would remark that in all probability the obstinate constipation predisposed to the location of the disease. The constant irritation of the hardened feces would tend to the deposition of the cancerous growth in this locality in a person predisposed to that disease. I have not seen constipation as a factor in the etiology of rectal carcinoma, but I think such a view plausible, and that we may infer the practical point—soluble bowels in the cancerously inclined.

May 24th, 1883.

III. THE VASCULAR SYSTEM.

1. *Heart with atheromatous coronary arteries, from a case of angina pectoris.*

Presented by Dr. S. F. HAZLEHURST.

Marion B., æt. 34, had suffered for several months from pain at the pit of the stomach, now and then occurring in paroxysms, attended with dyspnœa, the attacks gradually becoming more frequent and of increased severity, causing great distress.

The only noticeable symptom was an aortic murmur.

December 12. She returned to the dispensary, reporting herself as somewhat improved, which she attributed to a mass of small lumbricoid worms vomited that morning.

14th. She died.

Post-mortem, six hours after death.—Stomach first examined; somewhat congested in appearance. Heart rather more easily torn than usual; one of the aortic valves considerably ossified and bound back against the aortic wall. Coronary arteries almost obliterated. Aorta atheromatous.

December 22d, 1881.

2. *Cases of unusual cardiac disease.*

Presented by Dr. J. H. MUSSER.

CASE I. I saw the patient from whom this specimen was removed, the day previous to his death. He was so very ill that only a cursory examination of him could be made. There was general anasarca, *right* hydrothorax being especially noted. He was more or less cyanosed; suffered from extreme, constant orthopnœa; the kidneys secreted but a small amount. The impulse of the heart was diffused; the apex-beat in the sixth interspace outside of the nipple-line; loud systolic murmurs were noted over the mitral and tricuspid areas. The heart's action was irregular and rapid; the pulse rapid, small, and feeble. He was

58 years of age, had been an active business-man, a high liver, and a constant and immoderate drinker. The duration of the illness from the first cardiac manifestations was five years. Palpitation and dyspnoea were first noted. Œdema of the feet began two years ago. He never suffered from cardiac pain.

Post-mortem examination, forty-eight hours after death.—Rigor mortis marked; extremities and face blue; œdema of entire body; the right pleural cavity three-fourths filled with serum; the portion of lung not collapsed congested; left lung œdematous and congested. The heart was enlarged, weighing twenty ounces. The coronary arteries of both sides were tortuous and rigid from atheroma; the veins distended. The walls of the right side were about one-eighth of an inch in thickness, pale and flabby, and were very fatty along the septa and in small areas over the surface. The right cavity was enormously dilated; the tricuspid valves were healthy, but incompetent, admitting the fingers and thumb. The walls of the left ventricle averaged three-quarters of an inch in thickness; they were contracted and of a natural color. The mitral valves were healthy, but incompetent, admitting three fingers. Both auricles were dilated. The aorta was dilated and atheromatous throughout its course. The liver was enlarged and congested. The kidneys were characteristic of cyanotic induration, and the right contained a cyst as large as a walnut.

CASE II. Mr. D., æt. 83. With the exception of being an inveterate smoker, his habits were very good. Although always industrious, he has never been a hard worker. Forty-one years ago he had typhus fever followed by a chronic leg-ulcer. The past fifteen years he suffered from cardiac symptoms. At first palpitation, frequent sighing, and paroxysmal cardiac pains were noted. The pains increased in frequency and severity, but he never had true angina pectoris. The attacks of palpitation gradually became severe when quiet, as well as on exertion. For three years past he had suffered from attacks of dyspnoea at night, the desire for air arousing him from sleep; while in the mornings, on account of a sense of weakness about the heart and of inability to get his breath fully, he would not be able to leave his room for a full hour. Even then he could not recover himself without taking some warm drink. He noticed that as his leg-ulcer tended to heal, or when healed entirely, his cardiac symptoms were more severe; while the more abundantly the ulcer discharged, the better did he feel.

My attention was first called to the heart when treating him two years ago for erysipelas. The pulse was 60, though the temperature was 102°. I noted then a feeble pulse, a fair impulse, a weakened first and a weak second sound. He again came under my observation three weeks ago, suffering from pneumonia of the left apex. Along with the apex-consolidation, the remainder of each lung was congested. The pulse was slow, moderately full and feeble, the impulse somewhat lessened in force. When the congestion of the lungs became marked, and the respiration much increased, the pulse-rate became higher, at one time reaching 126. During the attack he had Stokes-Cheyne respiration at irregular intervals, and the previously-noted cardiac symptoms were marked. He died of heart-clot.

At the post-mortem examination the lungs were in the condition above detailed. The heart was rather under-sized for a large man, and it presented to the naked eye all the appearances of fatty degeneration. It was pale and flabby, and there was a large increase of fat, especially along the vessels. Its texture was soft, and it contained heart-clots. The other organs were healthy.

The first case is of interest on account of the disease of the coronary arteries without cardiac pain, and on account of the dilatation and degeneration being more marked on the right side than on the left, without any chronic pulmonary disease. The mouth of the right coronary artery is smaller than that of the left, but not more marked than in health. Although in the second case there were all the symptoms of a fatty heart, save that the impulse was readily detected and the pulse was not markedly feeble, and although the macroscopic appearances correspond to such a heart, yet I am not prepared to say that it is a fatty heart. A microscopical examination will be made and reported. The pains, of course, were only those of a false angina; and I am inclined to lay some stress on excessive smoking as an etiological factor in causing a neurosis, not only cardiac, but involving the respiratory nerves—a pneumogastric affection.

Dr. Musser said that these cases were of great interest, chiefly on account of their clinical histories being contradicted, as it were, by the autopsies. No microscopic examination had been as yet made to determine whether the "fatty" heart was really the subject of fatty degeneration or only of fatty infiltration. In the case with diseased coronary arteries no angina pectoris was noted, while in the other specimen *with healthy vessels* anginal pain had been a prominent

symptom. A microscopic examination of the heart-substance would perhaps explain this apparent discrepancy.

Dr. F. P. Henry thought it an interesting point that so much hypertrophy was coincident with such marked contraction of the nutrient arteries. He therefore would consider that the vascular stenosis was subsequent to the hypertrophy. For the production of cardiac hypertrophy at least two factors are essential, namely, a fair state of general nutrition and a patulous condition of the coronary orifices. Some time ago, Dr. Henry had shown two specimens to the Society illustrating these views. In one, the coronary arteries were enormously dilated, with great cardiac hypertrophy; while in the other, marked stenosis of those vessels was accompanied with a heart of normal size, the patient dying with extreme anginose symptoms. He would ask Dr. Musser whether in the patient with hypertrophied heart the pulse was slow or frequent.

Dr. Musser replied that the heart's action was rapid and intermittent.

Dr. Henry said that Prof. Alonzo Clark, of New York, had pointed out that in embolism of the coronary arteries a *suddenly slow* pulse was almost pathognomonic. Such an observation would naturally raise the question whether, in cases of a more gradual occlusion of these vessels, any aid to the diagnosis of the pathological condition could be obtained from a study of the pulse-rate.

January 12th, 1882.

3. *Fatty heart from a case of progressive pernicious or idiopathic anæmia.*

Presented by Dr. J. H. MUSSER.

As the notes of this case will be published elsewhere, with the Society's permission I shall only detail sufficient of them to show this to be a true example of that most interesting disease.

The patient was a female, æt. 46; menopause six months previous to illness; gave birth to a six-months' child twenty years ago; though twice married, never again became pregnant; failing in health and changing color for past three years, but in bed only four weeks; entire length of time in bed, seven weeks.

She was under my care three weeks. The marked symptoms and

features of the case were, briefly, as follows: Extreme anæmia; very little emaciation; earthy hue of skin; pallor of mucous membranes; irregular fever; acid perspirations; disturbed vision; subjective lights and noises; retinal hemorrhages; breathlessness; poor appetite; nausea and vomiting; constipation; slight enlargement of spleen; no enlarged glands; pulse rapid, small, and feeble; cardiac, arterial, and venous murmurs; no albuminuria; blood light-colored; corpuscles small, irregular, 715,000 red cells to a cubic millimetre, 15,000 white cells,—1 white to 47 red. Subsequently drowsiness and stupor set in, with delirium and coma, and death ensued.

Quantitative and qualitative analyses of the urine and blood were made, and will be published hereafter.

Post-mortem, twenty-four hours after death.—There were the usual appearances of the tissues, and I will only note, in addition to the appearances of the heart, that there were sub-peritoneal and pericardial ecchymoses; that the spleen was slightly enlarged, the liver fatty, the lymphatics normal, marrow of bone from radius and sternum healthy; brain not examined.

In pulmonary vein, black clot; in aorta and pulmonary artery, a soft yellow clot; in the venæ cavæ, soft red-brown clots, with black specks intermingled. The heart was pale, soft, flabby, and extremely fatty. The degeneration was more marked on the right side; along the septa and the vessels, in spots, there were areas of capillary injection. The right auricular wall was very thin, bands of muscular fibres being replaced by fat, while other bands seemed to be destroyed, so that parts of the wall were transparent, connective tissue forming the limiting membrane. The left side was similarly affected, but not in so intense a degree, there being only a diffuse yellowish discoloration. The papillary muscles of both cavities were markedly changed, the fatty change being shown by innumerable yellow dots. The right heart contained soft, semi-fluid, red-brown clots; the left, soft yellow clots. The aorta was a centimetre in width at the heart, and at the celiac axis admitted the little finger only.

I will report to the Society the results of a complete microscopical examination of all the organs.

Dr. Formad said that the microscopic examination of the bone-marrow revealed no such changes as he had observed in other cases of pernicious anæmia.

Dr. Tyson referred to Dr. Pepper's first paper on this disease, which he there had termed myelogenic leukæmia, wherein he showed that

this affection was always accompanied by medullary changes. In view of this fact, he was inclined to think that Dr. Musser's case should not be considered as a typical one of pernicious anæmia.

February 9th, 1882.

4. *Rupture of a fatty heart.*

Presented by Dr. J. M. BARTON.

I saw Mr. H. for the first time on January 27th. He complained of having had some pains in the stomach, but they had disappeared by the time of my arrival. Two days later he had a return of his pains: they were more severe than before, and were referred to the region of the diaphragm, with some shooting pains in the back. He was nauseated, and had vomited several times; his pulse was 148 to the minute, rather feeble, but regular and steady; it did not intermit, and its beats seemed to be all of equal force. The next morning he was much improved, the pain and other symptoms having been controlled by a small dose of morphia, and with the exception of a pulse of 104, fuller and stronger than the night previous, he did not present an abnormal symptom. He had eaten a fair breakfast, and was enjoying a pipe and the morning paper at the time of my visit. During the next two days he had occasional slight pains of the same character, not sufficiently severe to interfere with his appetite or sleep. On the third day, after an unusually hearty breakfast and dinner, he died suddenly, without premonitory symptoms.

On post-mortem examination, the cavity of the pericardium contained about five ounces of clotted blood. A rupture of the left ventricle at the apex was found.

The walls of the ventricle were pale in color, very soft and friable, and much thinned at the apex. The cavity was filled with fluid and clotted blood; the track of the rupture was sinuous and dilated in portions of its course; there were blood-stains under the visceral layer of the pericardium, about one inch around the point of rupture.

Dr. Neff said that he would like to know whether Dr. Barton had any reason to think that the rupture had occurred some time before death, and, if so, upon what he founded his opinion. He had made the post-mortem examination of a case in an old woman who had survived the rupture several days. The amount found in the pericardial sac had varied, in the cases examined by him, from a few drachms to

a pint or more. From these facts he did not think that it was the amount of fluid in the pericardial sac which caused death by interfering with the heart's action, since much larger quantities were often found in cases of pericarditis: where a large quantity of blood was effused, he was inclined to attribute death to cerebral anæmia; where a small quantity was at first poured out, the initial faintness was clearly due to shock, while if, later on, a large amount of blood found entrance into the pericardial sac, death then resulted from cerebral anæmia. All the cases examined by him showed what is usually found in the books,—viz., that the rupture was situated in the left ventricle, and usually at its middle and thickest part. A number of his cases had been produced by straining at stool.

Dr. Barton thought that the pericardiac pain, which also extended to the back, and the rapid pulse were produced by the permanent effusion of a small quantity of blood, which afterwards became augmented. He would call Dr. Neff's attention to the fact that the fluid effused in pericarditis usually collected *slowly*, while the amount suddenly poured out in cardiac rupture was often large, when compression of the organ was inevitable.

Dr. Neff thought that in such cases cerebral anæmia would occur before pressure could take place.

Dr. S. W. Gross thought that the amount usually present, from six to sixteen ounces of blood, was not enough to induce cerebral anæmia.

Dr. F. P. Henry thought that cerebral anæmia might well be excluded from the consideration of the causes of death in cases of rupture of the heart. No one cause was solely operative, three at least being concerned. These were—1, nervous shock, general and local; 2, interference with the heart's action from the presence of a foreign body suddenly introduced into the pericardial sac; and, 3, interference with the heart's action from division of its muscular layers. These three causes, he thought, were amply sufficient to account for death in such cases.

February 23d, 1882.

5. *Femoral artery from a case of amputation at the hip-joint.*

Presented by Dr. NANCREDE.

John A., æt. 21 years, a Swede, arrived in this port 4 A. M. February 22, 1882. While shifting the tow-rope, the rope slipped from the bitts, and, the tug steaming rapidly ahead, his left thigh was caught

in the bight of a seven-inch hawser, which produced a compound comminuted fracture of the upper third of the thigh, with rupture of the inner coats of the superficial femoral artery. The left testicle had also been torn out of the scrotum, and had slipped up under the skin of the groin. My resident, Dr. Neilson, promptly etherized the patient, replaced the testicle, suturing the scrotum, and awaited my arrival. I saw him about 12.15 P. M., and found him in an astonishingly good condition, although suffering considerable pain. The limb was evidently hopelessly disorganized in its upper third, nothing but the skin apparently being left in front and on either side. A large part of this detached skin was already dry and horny, looking like that of a cadaver after the removal of the epithelium permits drying of the integument. The superficial femoral could be felt pulsating strongly to about the upper opening of the canal through the adductor magnus, when all trace of it was lost. The limb from this point down was cold and tallowy-looking, with no trace of circulation in the popliteal or either tibial. Contusion extended up a little above Poupart's ligament in front, and over the back and outer part of the buttock.

Death being inevitable if left unrelieved in this condition, I first tied the femoral, and then, after applying an extemporized abdominal tourniquet, which was practically valueless, I disarticulated the limb, consuming probably not more than a minute in the operation. On account of the shortness of the fragment of the femur, its disarticulation took longer than usual. Owing to the promptness and efficiency of my friend Dr. Knight and my medical colleague Dr. Bennett, who happened to be present, and the skill of my resident, Dr. Neilson, aided by Dr. Watson, not more than five or six fluid-ounces of blood were lost. The patient bore the operation singularly well at first, but about half an hour after its completion he suddenly vomited profusely, the pulse ceased at the wrist, and it was only after two hours' work with whiskey, turpentine enemata, hypodermic ether injections, sinapisms over the heart, etc., that he reacted. This morning his condition was fair, with a warm skin, pulse about 120, of fair volume, skin-flaps sloughing in one or two places. He has vomited twice since the operation, but not for some hours. Part of the flaps are apparently united; there is but little oozing.

February 23d, 1882.

6. *Severe valvular lesions of the heart, complicated by acute intra-pleuro-pericarditis, with abundant serous effusion and peritonitis.*

Presented by Dr. J. T. ESKRIDGE.

J. B., æt. 22, German, working the last six months in a sugar refinery, was admitted to St. Mary's Hospital January 25 of the present year. His occupation had always been such as exposed him to the vicissitudes of the weather, but he had enjoyed what he considered perfect health until two years ago, when he suffered from pains in his knees and ankle-joints, but at this time his ailment was not sufficient to confine him to the house. Two and a half months before his admission into the hospital, weakness and pain in the region of the heart compelled him to cease work. He was anæmic, and his expression was that of anxiety. His temperature was 100°; pulse, 96, jerking and receding; respirations, 20 per minute. His urine was acid, scanty, and high-colored, but free from albumen. His lungs were apparently healthy, and his appetite fair. The principal and nearly the only thing complained of was pain in the left breast, which often extended to the shoulder and arm of the corresponding side.

The impulse of the heart was strong and forcible. The apex-beat could be felt in the sixth and seventh interspaces on a vertical line drawn midway between the left nipple and anterior border of the left axilla. Two murmurs, with their seats of intensity at or near the aortic orifice, could be heard. One occurred with the systole and the other with the diastole of the heart. The other orifices of the heart were, as far as I could determine, free from disease. Until February 1, nearly a week after my first examination, he seemed to be improving under treatment, and at this time suffered but little from his heart-trouble.

February 2 he complained of slight pain and an oppressed feeling in the region of the heart. I found that the area of dulness extended about one inch below the apex of the heart, whose impulse could now be felt in the fifth interspace. The dulness was pyramidal in shape, and extended to the right of the right border of the sternum. Over the breast-bone, on a line with the third costal cartilage, a to-and-fro friction-fremitus, synchronous with the heart's action, was heard. By firm pressure with the stethoscope its intensity could be increased at will. The friction-sound did not seem to be connected with respi-

ration, and whether or not it was loudest during a full inspiration was not observed. At all events, a pericarditis with effusion was diagnosed. Moreover, at this examination a mitral murmur could be distinctly heard in the left axilla, and faintly posteriorly at the lower angle of the left scapula. The pleuræ and lungs were examined, but no further trouble was detected.

It is but proper to state here that I did not see the patient any more during life, and that to Dr. Strittmatter, who carefully watched and cared for him, I am indebted for the remaining notes of his clinical history.

3d. Blistering, alkalies, and moderate doses of anodynes had failed to relieve the pain and distress in the cardiac region. By night the patient's suffering had greatly increased.

4th. He was seized with intense lancinating pain in the region of the heart, in each side of the chest, and in the abdomen. The respirations soon became hurried, short, and gasping. His countenance had a pinched expression, his face was pale, and his body bathed in perspiration. Dry cups and blisters gave but little relief. Morphia and atropia were administered hypodermically. Slight improvement then seemed to take place, and he slept about half an hour. He made no improvement during the night, and about ten o'clock on the morning of February 5, while being turned on the left side for a few minutes for the purpose of giving an enema, he gasped for breath, screamed, and fell back pulseless, respiration continuing about one minute after the pulse had ceased, during which time no heart-sounds could be heard.

Autopsy.—The examination was made about twenty-four hours after death by Dr. Strittmatter and myself, and was restricted to the chest and abdomen.

Chest.—The right pleural cavity was distended to its utmost with a serous effusion. Adhesions binding the right pleura to the pericardium and anterior portion of the chest were found. Only a trace of lymph was present in the effusion. The right lung was pushed upward and backward, and its lower lobe was in the second stage of pneumonia. The left pleura was adherent to the pericardium and the lower antero-lateral portions of the chest. Only a few ounces of serous effusion were in the pleural cavity of the left side. The left lung was intensely congested, but not inflamed.

The external surface of the pericardium was adherent to everything that lay in contact with it. Its surface was covered with lymph, and

its bloodvessels were prominently engorged. The visceral layer of the sac showed no evidences of inflammation, but several ounces of serous effusion were found in the pericardium. The heart was very much enlarged, the increase in size being due principally to enlargement of the left ventricle, which was found to be dilated and hypertrophied. All the valves, save those at the aortic orifice, were in an apparently normal condition. The aortic orifice was constricted by ossification and thickening of all its valvelets, thus giving rise to the systolic murmur. Bony vegetations half an inch long were attached to the apex of one of the valvelets, which must have played to and fro with the current of blood, allowed regurgitation to take place, and added intensity and roughness to both murmurs. No evidence of recent endocarditis was detected. The thoracic aorta was in a good condition. No cause beyond a dilatation of the left ventricle was found for the systolic murmur heard at the mitral orifice.

Abdomen.—Abundant evidences of peritonitis were found in the posterior portion of the abdominal cavity, where considerable serum and lymph were present, and numerous adhesions had taken place. The layers of peritoneum anterior to the bowels were nearly normal in appearance.

The kidneys were intensely red and apparently contracted. The remaining organs were apparently not implicated in the inflammatory process.

To sum up the pathological condition: The left ventricle of the heart was dilated and hypertrophied; the aortic orifice was roughened and constricted, and its valves, the seat of bony vegetations, were insufficient; the external surface of the pericardium was the seat of old and recent inflammation, and the sac was intimately adherent to the adjacent structures and contained several ounces of fluid; both pleuræ were adherent and contained abnormal quantities of fluid, the right being filled; both lungs were congested, and the lower lobe of the right was inflamed; there was peritonitis with its products; and the kidneys were contracted and congested.

Remarks.—The history of the joint-affection, as well as the condition of the urine in his last illness, points to rheumatism as the origin and nature of his trouble. It is instructive to observe the extensive heart-lesion, which probably began years ago, when he had so mild an attack of rheumatism of the joints that he was not kept in-doors. This case, with the numerous ones which have preceded it, should impress upon us the importance of a thorough examination of the heart in

every one suffering from the disease. The cardiac disease, as shown by the valvular ossification and the subsequent ventricular hypertrophy, was not of recent date. The cirrhotic condition of the kidneys followed the diseased state of the heart, rather than acted as a cause of the latter, which is probably more commonly the case. Can any one doubt that the healthy action of the heart would have been less impaired had the cardiac disease been detected during the first appearance of rheumatism and the patient confined strictly to bed? Much of the inflammation around the heart dated back at least two and a half months prior to his admission into the hospital, and was the cause of his distress at that time, which compelled him to stop work. Peritonitis is referred to by most authors on the practice of medicine as being one of the rarest of the internal complications of rheumatism. Dr. Flint, with a ripe experience, has met with only two cases. So diffuse an inflammation of the internal organs as here represented, associated with rheumatism, or more properly of a rheumatic origin, must indeed be exceedingly rare. I cannot find a similar case in the published Transactions of the Society.

A few words in regard to the diagnosis at the time I last saw him. At that time there were no evidences of pleurisy, but there were undoubted signs of pericardial effusion, the presence of which the post-mortem examination verified. Further, a double friction-murmur, more or less creaking in character, having an intensity that could be decidedly increased by firm pressure with the stethoscope, was heard over the base of the heart, above the seat of the effusion. No adventitious sounds were heard over any other portion of the chest except the præcordial region, save those of an endocardial origin. In the absence of all symptoms and physical signs of pneumonia and pleurisy, and with the presence of endocardial trouble and signs of pericardial disease, as evinced by a limited to-and-fro friction-fremitus associated with effusion in the pericardium, I felt that I could not doubt the presence of a pericarditis; yet the autopsy showed that there was no pericarditis proper. One resource in the physical diagnosis of pericarditis I omitted to employ, as I felt sure that a diagnosis in this case was easy. The point I refer to is the relation of the friction-sound to respiration. Intra-pleuro-pericardial friction-murmurs should be most distinct during inspiration, and become diminished or absent during expiration. In this patient two friction-sounds were heard with every cardiac beat, regardless of the respiratory act. Whether or not the fremitus was more distinct during inspiration than during expiration I

am unable to say, as I considered the diagnosis of pericarditis so certain that I did not endeavor to differentiate the supposed disease from that which most nearly resembles it. My error in this instance teaches me that all methods (no matter how unimportant they may appear) available in making a diagnosis of obscure diseases should be employed. They will at least corroborate more certain means.

When the man was first admitted, his temperature (100° F.) was about what we should expect to find in a slow form of inflammation. Such an inflammatory process was undoubtedly going on around the heart; but, as the hypertrophied condition of the organ showed the valvular trouble to be an old one, as there were no physical evidences of pleural or pericardial trouble, and as he apparently improved under treatment, and persisted in going about the wards of the hospital, the recumbent posture was not insisted upon. In the light of the autopsy, it seems probable that if he had been kept in bed during treatment acute inflammatory symptoms would have been less likely to result. In a person suffering from valvular lesions of the heart, especially when the cardiac trouble has a rheumatic origin, a temperature persisting above the norm and associated with oppression or uneasy feelings around the heart will in the future be a sufficient indication for me to enjoin absolute rest in bed for my patient, unless there exists sufficient rheumatic joint-trouble to account for the slight elevation of the body-heat.

Finally, I consider myself fortunate that I did not see the man during the last twenty-four hours of his life, when intense dyspnoea was the paramount symptom. Having made a previous diagnosis of pericarditis with effusion, there being no pleurisy, had I seen him gasping for breath the first impulse would have been to relieve his sufferings by aspirating the pericardium without further examination of the chest. With great enlargement of the left ventricle, a completely distended right pleura pushing the heart well over to the left side, and effusion in the left pleura pressing the pericardium, which contained only a few ounces (probably five or six) of fluid, in close contact with the heart, a plunge of the trocar could scarcely have relieved the pericardium without piercing the heart. It is not likely that any amount of surgical interference would have saved life in this case, but, had the true condition been recognized, tapping the right pleural cavity would have been the proper procedure. I have discussed this point at some length, because the fact is here illustrated that there are cases of sudden dyspnoea associated with pericardial effusion, for the relief

of which a surgical operation based on a diagnosis made only a few hours before would be liable to be followed by results of the gravest character.

March 31st, 1882.

7. *Great hypertrophy of the heart, due to severe valvular lesions.*

Presented by Dr. M. O'HARA.

J. H., male, æt. 30 years, laborer, contracted syphilis when 18 years of age. Health good until within past three years, when he began to suffer from pains in the left breast, palpitation of the heart, and slight dyspnœa, after violent exertion. Gradually becoming worse and unable to work, he was admitted into St. Mary's Hospital in June 1882. He then complained of great pain over the upper part of the sternum, in the left arm and shoulder. He lost considerably in weight, his appetite was poor, and albumen was present in small proportion in his urine, although casts were absent. He had marked dyspnœa on admission, which increased rapidly until sleep could only be obtained in the upright position. There was no evidence of pulmonary trouble. With the forearms flexed to a right angle, the brachial arteries became prominent at each impulse of the heart; the pulsation of the carotids was wavy and prolonged; the temporals were tortuous and visibly pulsated; no retinal arterial pulsation was seen. Retinal venous pulse was marked, but no visible venous pulsation was detected elsewhere. The left præcordial region was specially prominent. The apex-beat was most distinct in the sixth and seventh interspaces on a perpendicular line running midway between the left nipple and anterior border of the left axilla. The heart's impulse was usually forcible and diffused, but at times it became weakened and wavy. In the second left intercostal space a systolic impulse was observed. The pulse varied from 80 to 120 per minute, struck the finger with considerable force, but at once lost most of its volume. All these phenomena were exaggerated by raising the hands above the head. The radial pulses were unequal, but the brachial arteries presented no differences. No hepatic pulsation was felt. The cardiac area of complete dulness was nearly twice its normal size, the increase being downward and to the left. Over the second right costo-sternal articulation, the closure of the aortic valves was distinctly heard, and with this a slight diastolic murmur. A systolic murmur was also heard over the same spot. The systolic

murmur was nearly lost in the carotid and subclavian arteries, but the diastolic one remained distinct. On a line with the second costal cartilage, over the sternum, and to the left of this bone, the diastolic and systolic basic murmurs were most intense. Over the cartilage of the left fourth rib, the murmurs heard at the base of the heart were less distinct, or were obscured by the development of other murmurs. In this situation, a short, sharp, presystolic murmur, apparently prolonged with a systolic one, was detected. At the apex, and just above it, the systolic murmur became intensified, its blowing character aiding in differentiating it from the short, harsh murmurs which immediately preceded it. From the fourth to the seventh rib, and from the sternum to a point about five or six inches to the left, a diastolic murmur of considerable intensity was heard. The character of the diastolic murmur over this area was different from that presented by the second sound murmur at the base of the heart, and its intensity was much greater. Posteriorly at the lower angle of the left scapula, a blowing systolic murmur, entirely deprived of its harsh presystolic complication, was heard. Dr. Eskridge thought the physical signs justified him in venturing the diagnosis of constriction and regurgitation at the mitral orifice, a rare form of mitral regurgitation, produced by the inability of one of the aortic semilunar valves to close while the others acted properly; great hypertrophic dilatation of the left ventricle, and to a less extent of the left auricle; and aneurismal dilatation of one of the great vessels, probably of the pulmonary artery near its origin from the heart. The patient rapidly sank. Œdema of the feet, lower portion of the trunk and lower extremities, with hydrops pericardii, pleuræ and of the peritoneum developed. He died, exhausted, in August, nearly two months after admission to the hospital.

Sectio cadaveris.—Brain: Some venous congestion of the pia mater. The brain substance and the ganglia were nearly normal in appearance. Thorax: The pericardium was nearly filled with serum. The only evidences of inflammation were a few patches of recent lymph at the left anterior upper portion of the sac. The heart weighed eighteen ounces, the increased weight being chiefly due to eccentric hypertrophy of the left auricle and ventricle. The right ventricle was dilated, with slightly thickened walls. The aortic valves were insufficient and thickened. The posterior leaflet was normal in shape, but the others curled upon themselves on the aortic side of the orifice. The stenosis was slight. The mitral orifice was button-hole shaped, and the valves failed to close, on account of calcareous deposits in

their tissues, this degeneration also involving the inner surface of the left side of the auricle contiguous to the valves. The auricular surface of the valves was fairly smooth, but in the ventricle just beyond the valves, and attached to them, hung a bony substance about one-eighth of an inch in diameter. The left auricular appendix was much hypertrophied. The valves at the tricuspid and pulmonary orifices were normal. The pulmonary artery was considerably dilated. The pleural cavities contained several ounces of serum. There were old adhesions at the apex of the right lung posteriorly, and at the same spot of the left lung anteriorly. Several patches of recent lymph were also found at the lower part of the left pleural cavity. Abdomen: The peritoneal cavity contained considerable serum, and its veins were engorged. The liver was enlarged, with distended veins. The kidneys were highly congested. The spleen was double its normal size, and softened.

November 23d, 1882.

8. *Dilatation and atheroma of the pulmonary artery, with an opening through the interventricular septum.*

Presented by Dr. E. T. BRUEN.

Examination of the Heart.—Left side: Slight ventricular hypertrophy; mitral valves somewhat thickened at the margins, with roughening of their auricular aspect; valves competent; the left auricle is normal, as are also the aorta and the aortic valves. Examination of the right side is of most interest. Two of the semilunar leaflets at the mouth of the pulmonary artery are nearly destroyed by atheromatous changes, while the third segment is much thickened and projects as a leaf-like fold, roughening the mouth of the pulmonary artery. This vessel is dilated to twice its normal size, forming really an aneurismal dilatation. The vessel walls are covered with a fringe of vegetations, of inflammatory origin, or due to atheromatous changes. The right auricle is very small and imperfectly developed, the bulk of its cavity being formed by the auricular appendix. The tricuspid valves are much thickened, but are probably competent. Between the two ventricles is an orifice large enough to admit the forefinger. It is directly beneath one of the two tricuspid leaflets, and is lined with endocardium, and must have allowed a free interchange between the blood of the two ventricles. The walls of the right ventricle are thinned and its cavity somewhat

dilated. Dr. Bruen said that this case was interesting, (1) because perforation of the ventricle or septum is often congenital and dependent on obstruction of the orifice of the pulmonary artery, the perforation being due to the pressure of blood within the replete right ventricle. This pressure causes an arrest in the development of the ventricular septum. The pathology of the present case probably is as given above, but there was no pulmonary artery obstruction. A similar case is recorded in the *Medico-Chirurg. Trans.*; vol. xv., by Fletcher. (2) There was no cyanosis. Cyanosis is usually dependent on a deficiency of cardiac evolution, or else on retarded evolution of the pulmonary artery or aorta. As a consequence there is deficient cardiac power to carry on the circulation; or the pulmonary artery or the aorta is narrower than normal, so that in any of these conditions venous repletion results and cyanosis; mixture of the venous and arterial blood is not, then, a usual cause of cyanosis, although it may be a factor. Walsh says, "Grant that perforation of the ventricular septum coexists with constriction of the pulmonary orifice, and cyanosis seems to become a certainty." In our case there is an example of incomplete development of the ventricular septum and deficient development of the right auricle, without cyanosis. (3) Pulmonary artery disease is consistent with a fair amount of general health, and compensation by the right heart may occur, just as in cases of aortic disease. (4) Descriptions of pulmonary artery disease call attention to bronchitis, pneumonia, and hydrothorax, as sequential states. In our case no such complications were present until just before death, when the patient finally succumbed to congestion of the lungs added to the cardiac state. (5) The aneurism of the pulmonary artery formed a pulsating tumor on the left side of the sternum, between the second and fourth ribs, extending outward from the border of the sternum and including an area covered by a trade dollar. (6) Over the tumor a post-diastolic and a pre-systolic, bruit-like murmur could be heard, at a point between the second and fourth ribs, while close to their junction with the sternum a hoarse systolic murmur could be heard. The bruit was localized; the heart systolic murmur was carried out into the entire arterial system.

Dr. Bruen then detailed at length the differential diagnosis of these murmurs. During life dilatation of the pulmonary artery, with mitral obstruction, had been the diagnosis. The patient was a woman, aged 24 years, a syphilitic, and was under observation from November, 1878, to July, 1882.

Dr. Shakespeare said that he had been struck with one point of great interest in connection with inflammation of the lining coats of the pulmonary artery, as evinced by the vegetations. These growths are very rarely found in the venous current. He had certainly never seen any other specimen, although he did not doubt that some had been seen or reported by other observers. Arterial blood seemed a requisite for the evolution of such diseased action. Evidently the site of the perforation being just below the aortic and pulmonary valves, brought about just this necessary pre-requisite, viz., abundance of arterial blood within a vessel which normally carries venous blood.

Dr. Wilson called attention to the evident relation between the incomplete ventricular septum and the condition of the pulmonary artery, which is greatly dilated and atheromatous, and presents the appearances often met with in the aorta, but very rarely in this vessel. The walls of the right heart are relatively thickened. This fact, together with the position of the opening in the interventricular wall, which favors the flow of the blood from the left ventricle toward the pulmonary artery, renders it probably almost certain that the more forcible contraction of the left heart has constantly forced a portion of its arterial blood into the right heart, thus increasing the current entering the pulmonary artery and occasioning, 1st, hypertrophy of the right ventricle, and 2d, a subacute inflammatory process in the pulmonary artery itself, in consequence of the increased volume and force of the blood current. Dr. Shakespeare's observation that such growths as are here seen require for their existence arterial blood is in accordance with this view.

November 23d, 1882.

9. *A case of mitral obstruction, with sequential lesions.*

Presented by Dr. E. T. BRUEN.

I submit this specimen because it favorably illustrates the lesions and the sequential changes in the different chambers of the heart. The auriculo-ventricular opening of the left heart is nearly occluded by an epiglottic-shaped enlargement of one of the leaflets of the mitral valve. The valve is very much thickened, and the focus of a considerable calcareous deposit. The orifice during life permitted a reflux of blood from the ventricle into the auricle. The left auricle is dilated and hypertrophied so that its cavity is about twice as large as

normal. The right ventricle is very much dilated, the walls of this cavity are less than half the normal thickness, and the ventricle must have had during life twice its physiological capacity. The tricuspid valves were insufficient on account of the dilatation. The considerable enlargement of the left auricle and right ventricle occasioned during life a broadening of the area of dulness on the level of the third and fourth ribs, viz: the apex of the cardiac triangle. It also produced a decided increase in the area of the cardiac dulness to the right of the median line of the sternum. In children, the heart with similar enlargement encroaches upon the left pleural cavity to such an extent that the physiological inflation of the left lung cannot occur. Bronchial breathing is produced, audible posteriorly, while anteriorly, below the second interspace, no respiratory murmur is audible. In these cases, when the complication of bronchitis occurs, the physical signs suggest a pleural effusion. Enlargement of the right ventricle, both in children and adults; causes a pronounced impulse at the epigastrium, and occasions serious pain and inconvenience. The murmur heard during life in the case from which my specimen was taken indicated this lesion, both presystolic and systolic murmurs being audible. The second sound at the pulmonary artery cartilage was also much accentuated, owing to the repletion of that vessel with blood. The first sound over the right ventricle was very clear and distinct, as is common in these cases, but the first sound at the apex was obscured by the murmur. The patient, from whose body these specimens were removed, was a woman aged 45 years, who had been subject to heart disease since she was twenty years old. The immediate cause of death was pulmonary repletion with blood, which induced right-heart failure. Compared with mitral regurgitation, this mode of death illustrates a feature of the chemical pathology of mitral obstruction. In mitral regurgitation death occurs with heart failure, but usually after serious dropsy. In mitral obstruction dropsy is not so prominent a symptom, but the pulmonary engorgement prevents a perfect supply of blood to the aorta. The right ventricle failure in cases of mitral obstruction brings about death in the same manner as the left ventricle failure does in cases of aortic obstruction.

December 14th, 1882.

10. *Pericardial effusion and adhesion of the pericardium to the apex of the heart, mistaken for heart rupture.*

Presented by Dr. J. T. ESKRIDGE.

Dr. Eskridge said that in this case the physician making the autopsy actually considered the specimen to be one of heart rupture. The patient was an athletic young man, and perfectly well until a few days before he sought medical advice. He was under treatment for only 24 hours, suffering from cardiac pain and great prostration. He died suddenly and unexpectedly when no one was near him. The attending physician, who made the post-mortem examination with no professional assistance, reported effusion in both pleural cavities, the pericardium distended with thin, non-coagulated blood, and a rupture of the left ventricle. Dr. Eskridge said that a careful examination of the heart, pericardium, adjacent glands, and portions of the larger bronchi showed marked evidences of pericarditis and pleuro-pericarditis. The pericardium was adherent to the lower third of the heart, but the adhesions were recent and easily severed. The heart was not much enlarged; its valves were nearly normal, and the muscle firm. No rupture was found. He believed that the case was one of pleurisy and pericarditis with effusion, death taking place suddenly from mechanical interference with the heart and lungs. He thought that the most plausible explanation of the doctor's mistake in calling it a case of cardiac rupture was, that when severing the bloodvessels around the heart, blood flowed into the pericardium and mingled with the serous effusion. He did not think that a firm, non-fatty heart could rupture itself by its own contractions. If the pericardium was filled with effusion, in that instance it taught a lesson of far more practical value than a case of cardiac rupture under similar circumstances would. It was evident, if the pericardium should be attached to the apex of the heart in a case of pericardial effusion, in which operative interference was determined upon to free the heart's action, a thrust of the trocar into the pericardium would greatly endanger the ventricular walls.

December 14th, 1882.

11. *Case of stab-wound of the pericardium, diaphragm, and liver.*

Presented by Dr. STRITTMATTER.

The patient from whom this specimen was taken was admitted into the Surgical wards of St. Mary's Hospital, July 16 of this year, during the service of Dr. Mears, to whose kindness I am indebted for being allowed to present it.

Ludwig H—, aged 62, German, laborer, came to America about three months prior to his coming into the hospital. He resided in Brooklyn. Two days prior to his admission he left Brooklyn and came to Philadelphia, laboring under the delusion that he had outraged a young woman, and that he was about to be arrested for his crime. He spent the night and following day with friends. In the evening, after writing a very intelligible and affectionate letter to his wife and children, he drew a large single-bladed clasp-knife and stabbed himself several times in the right chest, holding the knife in his right hand. He remained standing, and struggled vigorously against the interference of his friends. He was brought to the hospital at once, and on admission was found to be much excited, trying to run away, being under the impression that he was to be punished for his supposed crime. There was not much shock, his face having rather the appearance of flush than of pallor. Examination revealed two wounds in the integument of the chest. One was situated over the cartilage of the sixth rib of the right side, about an inch and a half from the border of the sternum, and was about two inches in length, its depth being limited by the costal cartilage. The other wound was about an inch lower, and one-half inch from the right border of the sternum, severing the cartilages of the seventh and eighth rib from it. Closer examination revealed the fact that at this point there were two openings through the costal cartilage, about a line apart externally; the outer being in a direction downward, outward, and backward; the inner in a direction inward, downward, and backward. Through these openings air passed occasionally, when the patient made violent respiratory efforts, causing a high-pitched, sucking, blowing sound. Little bleeding took place, and that little from the integument only.

Physical examination of the chest revealed normal percussion resonance over the left side of the chest, including the cardiac area, and over the upper portion of the right side; increased resonance was noted over the lower portion, and, on heavy percussion, a sort of "cracked-

pot" sound. On auscultation, the normal vesicular murmur was heard over the left lung, diminished breathing sounds over the upper portion of the right lung, with absence of respiratory sounds in the lower portion. Heart sounds were distinct, but lessened in intensity, especially the first, which was rapid, and at times irregular. The man was very nervous, but on the whole his nervousness and anxiety seemed to be due more to his fear of being arrested for his supposed crime than to his injuries. There were no symptoms of internal hemorrhage. Pulse 112° ; respirations 42, and shallow. When the head and shoulders were raised, and he took a full inspiration, a peculiar high-pitched blowing sound was heard occasionally, just a little to the left and below the ensiform cartilage during both inspiration and expiration. This sound was not constant, and was peculiar in character, being unlike the sounds produced by gas in the stomach or bowels.

Antiseptic dressings were used, and morphia was given, which produced but little sleep during the night. He gave no manifestations of anxiety that could be attributed to his wounds, and complained only of a burning sensation under the sternum. On the following morning the patient was much calmer, answered all questions intelligently, and complained only of pain during inspiration. His breathing seemed freer and easier during the night, but his abdomen had grown tympanitic. All the other physical signs remained the same. Pulse 92, moderately full and strong; respirations 32; expiration was attended with a forcing effort and groan; temperature 101.6° . Evening: physical signs are unchanged; pulse 88; respirations 24, and easier than in morning; temperature 101.6° . During the night the patient grew very restless, slept little, and in the morning he complained of sharp cutting pains all over the chest with each inspiration. His countenance had assumed a sharp, pinched expression, and he manifested considerable anxiety.

On auscultation, friction sounds were heard over the pulmonary area from the apex to the base on the left side, and from the apex to the fourth rib on the right side. Friction sounds also accompanied the movements of the heart. The pulse was 100, full and hard. The temperature had risen from 101.6° on the preceding day, to 103.2° ; respirations 48; inspiration was rapid and jerky, terminating abruptly, followed in a few seconds by expiration, which was forced, prolonged, and attended by a groan. The high-pitched, blowing sound was still heard under the ensiform cartilage, but not constantly. The abdomen was tympanitic, but not painful to the touch. These symptoms con-

tinued during the day. In the evening the breathing appeared to be somewhat easier, although no effusion could be discovered; pulse 120, respirations 44, temperature 103.2° . During the night the patient grew much calmer, and slept some after 12 o'clock. On the following day he appeared much better, did not complain of pain, and took some food. Physical examination, while the patient was lying on his back, revealed flatness on percussion from the back to about midway between it and the plane of the anterior surface of the chest on the right side; the dulness was not quite so extensive on the left side. Over this area vocal fremitus and resonance were feeble, breathing sounds absent. The friction sounds over the cardiac area had given place to dulness on percussion, and diminished heart sounds. The area of dulness could not be mapped out accurately on account of the pain produced by percussion. Friction sounds were still heard at the left apex. Blowing sound under the sternum was no longer heard. The abdomen was quite tympanitic, no part gave dulness on percussion, and it was not painful on pressure. The evening of the same day the breathing was considerably embarrassed, but not painful, and was unattended by the groan of the previous day. The right side was found quite flat on percussion; breathing and friction sounds were not heard. The left side was dull over a greater area than in the morning; the friction and breathing sounds had also disappeared. The heart sounds were scarcely audible, especially the first. Pulse 120, irregular; occasionally intermitting; respirations 40; temperature 101.8° .

During the night the patient was restless, tossed about a great deal, and at times made attempts to sit up. In the morning (20th), he appeared exhausted; his countenance was pinched and pale. His forehead was covered with perspiration; his anxiety was great, and he spoke hurriedly, and in short sentences. The pulse was 126, feeble, and occasionally intermitting; respirations shallow, 40 per minute, and attended with considerable effort; temperature 102.8° .

He sank occasionally into a condition of semi-unconsciousness, attended by low muttering delirium, from which, however, he was aroused by the slightest noise. He sank rapidly, his pulse growing more feeble and rapid, and respirations more hurried and shallow until 2 P. M. of the 20th, when he died. In the few hours preceding death his temperature rose steadily, and at death reached 105.5° Fahr.

Autopsy sixteen hours after death.—*Brain:* Pia mater adherent; thickened in part, and opaque, especially on either side of the vessels, which were filled with dark blood over the upper convex sur-

face of the left hemisphere. A slight amount of serous effusion was found in the subarachnoid space. The pia mater of the right hemisphere was slightly adherent, but could be taken away in considerable patches without tearing; the vessels were in a less turgid condition. The ventricles contained little over the normal amount of fluid.

Chest: On raising the sternum and costal cartilages, the right pleural cavity was found filled with thick fatty-looking effusion, with bands extending between the parietal and pulmonary pleura. At the apex the effusion was firmer and served to bind the pleural surfaces together. The anterior portion of the parietal pleura was covered with soft effusion of a grayish-yellow color to the depth of one-quarter of an inch. The posterior portion of the lower lobe of the lung sank in water. On the right side the effusion only half filled the pleuritic cavity, and both surfaces of the pleura were covered with false membrane fully one-quarter of an inch thick. At the apex the two surfaces were united by bands of lymph. No portion of the left lung would sink in water. The pericardium was distended with fluid, and lined with an abundant layer of lymph. The heart was covered by a similar layer, and the two surfaces connected by numerous bands. The left ventricle contained a chicken-fat clot, extending about six inches into the aorta; the right ventricle was filled with blood, a small clot extending into the pulmonary artery.

While there was evidence of only two penetrating thrusts of the knife externally, several thrusts must have been made without completely withdrawing the knife from the external opening, as three distinct openings were found in the diaphragm, extending into the liver, to the right of the suspensory ligament of that organ, and one entering the pericardial sac, about an inch above its attachment to the diaphragm, extending through the attached portion, the diaphragm, and one inch into the left lobe of the liver. The other three wounds in the liver were not so deep, being one-half, one-quarter, and one-sixth of an inch in depth.

Abdominal Cavity: There were no traces of peritonitis, except about two ounces of serous effusion, which probably escaped from the pericardial sac. The edges of the wounds in the liver were not separated, were un-united by any exudation, and presented sharply defined edges slightly tinged of a brownish-yellow hue. On section, this discoloration was found to extend for about an inch in every direction from the wounds into the substance of the gland. The organ weighed sixty-two ounces, and was not appreciably enlarged or altered in

shape. The spleen was enlarged, soft, and pulpy. The kidneys were normal. The intestines did not contain sufficient gas to account for the tympany during the last two days of life.

Remarks.—The points which have been of particular interest in the case are the wounds of the liver, without hemorrhage and peritonitis; and the origin of the peculiar blowing sound occasionally heard under the lower portion of the sternum.

Many cases of wounds of the liver have been reported, the patients living for days and even weeks without hemorrhage, and Dr. Otis states that out of one hundred and seventy-three cases of gunshot wounds of the liver occurring in the late war, there were thirty-two undoubted recoveries. All authorities, however, agree that there is always great danger of hemorrhage and peritonitis.

In this case the wounds were incised, and all sufficiently deep to cause bleeding, being one-sixth, one-quarter, and one-half of an inch in depth, while the last was fully one and a half inch in length. Still, there was very little shock, no hemorrhage diagnosed by symptoms, or verified by post-mortem, and not even a trace of inflammatory action in or about the wounds, only a brownish-yellow discoloration extending about an inch into the liver substance. During life there was no icteroid discoloration of the conjunctivæ or integument, and no itching, symptoms usually said to be found in wounds of the liver. The second point of interest is, the peculiar sound heard occasionally under the left lower portion of the sternum. In consulting the different authorities on wounds of the diaphragm, I have been unable to find any physical signs by which a wound in the diaphragm could be diagnosed during life, except those accompanying the protrusion of part of the abdominal viscera into the pleuritic cavity. In this case, the wounds were too small to allow any such protrusion, and even had they been larger, such an occurrence would have been prevented by the close contact of the diaphragm with the left lobe of the liver. In penetrating wounds of the diaphragm, unaccompanied by air in the pleuritic cavity, the production of any sound is impossible; but where a condition of pneumothorax exists, either from wound of the lung or from an opening in the chest wall not sufficiently large to allow a free escape of air during expiration, as was the case in my patient, I think it is quite possible, and even probable, that part of the air will be driven into the abdominal cavity, and that its passage must produce some sound. In this case, when the sound was first heard, it was supposed to be of intestinal origin. It was only occasionally heard,

several respirations taking place, during which no sound was heard, yet its constancy in site and character, its accompanying both inspiration and expiration, its greater intensity during expiration, and the development of tympany without tenderness of the abdomen on pressure, and the absence of other symptoms of peritonitis, led to the belief that the sound was produced by air passing in and out of the abdominal cavity. The disappearance of the sound when the right pleural cavity and pericardium were filled with fluid, and the openings were covered over and filled with masses of exuded lymph, served to strengthen this belief. I cannot find or think of a term that would fully describe the sound heard, and hence will refer it to my chief, Dr. Mears, whose attention it attracted on the second day after admission.

Remarks by Dr. Mears.—I was much interested in the above case, the clinical history of which, with the post-mortem appearances, has been so fully described by Dr. Strittmatter. During life the symptoms of wound of the diaphragm and of the liver were markedly absent, whilst those of injury of the pericardium and pleura developed as the interval, after the receipt of the wounds, increased. The external wounds gave little indication as to the direction taken by the knife after penetrating the thoracic cavity, and, as shown by the post-mortem examination, no information as to the extent of injury inflicted. The absence of symptoms of injury of the diaphragm may be explained by the fact that the wounds were in the tendinous portion of that muscle, and, being small, did not interfere to any great extent with its function in respiration. In injuries causing laceration of the muscular fibres attached to the ribs dyspnoea occurs as a prominent symptom by reason of the impairment of the respiratory duty of the muscle. Moreover, the symptoms may have been masked by those referred to the injury of the pericardium, as in wounds of both of the structures dyspnoea is a prominent symptom. The knife in one of the thrusts passed through both, and involved them in a common injury.

The only explanation I can offer of the production of the blowing, or rather suction sound, which was heard under the ensiform cartilage, is that it was occasioned by the passage of air during respiration through the openings in the diaphragm—the air entering primarily the lung cavity through the external wound. The fact that the air did not pass in and out of the external wound during the act of respiration afforded good evidence that the lung was not wounded.

The wounds of the liver were of such a character as to make little

or no impression beyond what might occur as the result of injury to the coverings and superficial portions. Puncture of the liver with the trocar is performed, at the present time, frequently with the view of evacuating fluids. Instances are reported in which no fluid has been found and no harm has been inflicted by the tapping. Extensive lacerations, the result of gunshot wounds or rupture, following falls, produce characteristic symptoms of shock and internal hemorrhage.

December 14th, 1882.

12. Ossification at the aortic orifice.

Presented by Dr. J. T. ESKRIDGE.

The specimen was sent him from a distance, and consisted of about one inch of the cardiac end of the thoracic aorta, the aortic semilunar valves, and the immediate portion of the heart. The specimen was removed from a man who, aged about seventy, had suffered a number of years from severe heart disease. The walls of the large arteries were thickened, rigid, and contained numerous deposits of inorganic matter. The left ventricle was enormously enlarged. He was unable to obtain any information with regard to the condition of the cardiac valves other than those of the aortic orifice.

Description of the specimen.—The aorta, where it surrounded the valves, for about half an inch in extent, was a hard, unyielding substance of fibrous tissue and calcified and ossified matter. The valves in several places were about one-fourth of an inch thick, and seemed to have been almost entirely transformed into bone-like material. They were rigid and immovable, and had almost completely cut off all communication between the heart and aorta. One of the leaflets, about three-fourths of an inch in all directions, with its vegetations, stretched across the aorta, lay against, and was apparently adherent to, the other segments of the valves, the latter being curled upon themselves. The central portion of the aorta was entirely occluded, and only two small openings, through which the blood could have escaped from the left ventricle, were seen between the valvular leaflets near their peripheral attachments. The larger of these holes admitted a flattened probe three mm. wide by one thick; the smaller was about two-thirds as large. Three other smaller orifices had existed, but these were obliterated before death by a thin, fibrous, transparent

membrane, which was still seen. The valves on the cardiac side were tolerably smooth, but on the aortic side they were very rough, one of the leaflets supporting a vegetation ten mm. long. One of the segments of the valves was adherent to the inner coat of the aorta for about half an inch in extent, the free end of the valve being folded upon itself and pointing toward the nearly closed aortic orifice. After macerating the specimen in water for forty-eight hours, the diseased valves still remained inflexible.

Drs. Tyson and Nancrede called in question the correctness of calling the disease *ossification* of the valves, as it was in reality a *calcification*.

Dr. Shakespeare concurred in this view, and thought that ossification rarely, if ever, occurred in this situation.

Dr. Eskridge said that Hayden¹ referred to bony deposits in the aorta and its valves as follows: Sir Dominic Corrigan exhibited before the Pathological Society of Dublin² the heart of a young woman in which the root of the aorta had undergone complete osteoid transformation; it was likewise greatly dilated, and the aortic valves had been rendered thereby inadequate. During the patient's last illness, a systolic murmur of metallic quality, appropriately designated a "trumpet-bruit," was audible at the base, and in the ascending aorta and carotid arteries; there was likewise a soft diastolic murmur. He regards a "trumpet-bruit" as absolutely diagnostic of bony deposit in the aorta, either in the form of a "rim of bone," or a "projection or tongue of bone." In the same paragraph Corrigan refers to Dr. Banks's specimen of a "tongue of bone" projecting into the aortic orifice.

March 8th, 1883.

13. *Case of aneurism of the arch of the aorta.*

Presented by Dr. GUY HINSDALE.

William G., a fireman, aged forty-four, was admitted to the Episcopal Hospital September 16, 1881, suffering from pneumonia of the right side, associated with signs of pleurisy. It was noticed at the time of his admission that there was a slight diastolic murmur heard

¹ Diseases of the Heart and Aorta, vol. ii. p. 839.

² See Proceedings, New Series, February, 1864.

at the aortic cartilage, and that no radial or ulnar pulse could be felt upon the left side.

The pneumonia pursued its course until it ended in death on the eleventh day after admission. Eighteen hours before death occurred, however, after an attack of severe pain along the course of the sciatic nerve, he suddenly became unconscious and his respiration puffing. His pupils were contracted, and his left arm rigid. His pulse became at once fuller and stronger than it had been since admission, there being ninety-six beats per minute. Fifteen hours before death the right side of the face was paralyzed. The radial pulse was smaller on the right side than on the left. The right foot was cold and the posterior tibial pulse behind the internal malleolus was just perceptible. Respirations were forty per minute and stertorous, sweat and urine profuse. Twelve hours before death, respirations 57, temperature 106.2° F.

The patient remained essentially in this condition until his death. At the post-mortem examination, upon opening the thoracic cavity, the base of the right lung was found in the second stage of pneumonia. There was hypostatic congestion of the left lung. But what at once threw light upon the later features of the case was the presence of an aneurism of the transverse portion of the arch of the aorta. The specimen shows a symmetrical dilatation of the vessel capable of receiving a large hen's egg. On examining the brain, clots, probably of embolic origin, were found in the right cerebral hemisphere, thus accounting for the paralysis which had been observed.

April 12th, 1883.

14. *Case of aneurism of the common and external iliac arteries.*

Presented by Dr. GUY HINSDALE.

Johann B., a farmer, aged twenty-six, was admitted to the Episcopal Hospital October 27, 1882. His previous health was good. He denied specific history, and his habits were good. He was able to work until one week previous to admission, when he began to have pain in the feet and legs shooting up the body to the head. His skin was yellowish, his tongue coated; his urine did not contain albumen. There was cedema of the feet, and it is worthy of note that this had occurred on the right side four months before admission. His spleen

was apparently enlarged ; his lungs presented nothing abnormal. Systolic and diastolic murmurs could be distinctly heard all over the cardiac area, but most clearly at the right second cartilage, and transmitted along the left subclavian and carotid vessels. The heart was considerably hypertrophied.

Six weeks later the attacks of pain in the right thigh and side, to which he had been subject ever since admission, became more frequent and severe. The murmurs at the aortic valve were systolic and diastolic, low, soft, and confused. A distinct thrill was now felt ; heart's impulse quick and trembling.

About one week before death a swelling occurred in the right parotid region, extending to the cheek and lower jaw. Subsequently a thin and purulent discharge took place from the ear, and the patient became unable to protrude his tongue. The œdema of the right leg returned and increased daily, extending up to the body, the whole leg being about three times the bulk of the other. The aneurism grew with great rapidity ; the distension of the vessel and the overlying structures gave rise to the greatest pain. The patient was unable to lie down and was always found in the sitting posture. The mind was clear up to the day of his death, which occurred two months after admission.

At the autopsy, upon opening the abdomen, the aneurism was found very prominent, occupying the right side of the abdominal cavity and extending from the concavity of the liver to Poupart's ligament, a distance of about nine inches. The aorta bifurcated at the upper border of the fourth lumbar vertebra, the tumor arising from the common iliac artery one inch from the aorta. Its whole anterior surface was covered by peritoneum. The right kidney was elevated, lying upon the tumor's upper surface, the ureter passing over the convexity of the tumor and to the inner side. The psoas muscle and crural nerves were displaced and stretched to the outer side of the aneurism. The femoral artery emerged from the tumor one inch above the origin of the profunda.

Upon opening the sac a second sac was found within. No rupture had taken place. The cavities of both sacs were found filled with clots in various stages of organization. The walls were leathery, and large flaps of organizing clots were found attached to them. There were also post-mortem coagulations.

The line of the blood current could be determined by the passage of a catheter. This was found both from above and below to skirt the

inner side of the tumor and to pass through a comparatively short channel in its interior. Three of the bodies of the lumbar vertebræ were slightly eroded. The heart was hypertrophied; its weight fifteen ounces. The mitral valves were normal, but the aortic valves showed exuberant vegetations which impeded their action and which were very friable.

The specimen presented embraces the heart, the aorta, the aneurism with the right kidney upon its upper surface, the right femoral and the left iliac arteries.

April 12th, 1883.

15. *Case of aneurism of the abdominal aorta and left common and external iliacs.*

Presented by Dr. GUY HINSDALE.

Mary K., aged thirty, was admitted to the Episcopal Hospital August 26, 1882. Her mother died in confinement, and her father of heart disease. She had had three children; her husband was a bartender, and had been treated for syphilis at a dispensary; but the patient herself denied specific history, nor were any evidences of it found. She had never been ill before.

Seven months previously she had pains in her left groin; these pains extended to the abdomen, and about five months previous to admission she noticed a small tumor in the hypogastrium which pulsated and became painful on pressure. Three months later her strength began to fail; she became delicate and lost flesh. For one month the tumor had been prominent. Pain then came on spontaneously at intervals, and she had backache. She became weak and took to her bed. Two weeks before admission the left leg and thigh became painful and felt benumbed and motion grew difficult, and for a time was lost. Meanwhile the tumor increased.

Upon admission she was weak. Her face showed anxiety and suffering and had a haggard and pinched look. The organs of digestion were not interfered with. Her lungs and heart were normal. On the right side of the abdomen a tumor was felt one inch above the umbilicus and extending from one inch to two and one-half inches from the median line, downwards two inches. It was painful on pressure, pulsated distinctly, and over it a distinct systolic bruit could be heard. A distinct notch separated this from a large hard mass in the left iliac

fossa, indistinctly pulsating, immovable, and not particularly painful on pressure. There was a faint bruit.

In the left thigh there was insensibility to touch over the distribution of the anterior crural nerve, while pain in the left knee was often so great as to deprive her of sleep.

The urine did not contain albumen.

At the end of two weeks sensation was diminished on the left side as high as the edge of the ribs and in the whole of the lower extremity, excepting the first and second toes; it was impaired over the buttock. Oedema of both legs occurred. The posterior tibial on the left side could scarcely be felt to pulsate.

September 13 the patient suddenly died. At the autopsy the abdomen was first opened. There was no blood in that cavity. Upon the left side in the iliac fossa there was a prominent tumor of the size of a cocoanut; over it lay the psoas muscle, which was spread out in the shape of a thin aponeurosis. The iliacus muscle was displaced. The psoas parvus was inserted into the tissue covering the tumor.

A fusiform dilatation of the aorta was then noted. The diameter of the aneurism was three times the normal diameter of the aorta. Commencing from a point seven inches above the bifurcation, it extended downwards nearly five inches.

A second aneurism occupied the left iliac fossa, involving the left common and external iliac arteries. It was about six inches in length and was filled with old fibrous, laminated clots. The latter aneurism had opened posteriorly behind the peritoneum and against the iliac bone. There had evidently been ruptures previous to the one which finally caused the patient's death.

There were found old as well as recent clots outside the aneurismal walls. There was no erosion of the bones. The femoral artery was normal, and no atheroma or evidences of syphilis observed, although the woman had been exposed to infection from a syphilitic husband.

April 12th, 1883.

16. *Case of extreme mitral stenosis, death resulting in a few months from sequential lesions without general dropsy.*

Presented by Dr. J. T. ESKRIDGE.

Charlie, æt. fifteen years, died in St. Mary's Hospital, December, 1882, during my term of service. In February of that year he first came to the hospital suffering from acute bronchitis. The attack ran

its course in a week or two, but the heart, during and after the seizure, was exceedingly irritable, frequently beating from 120 to 150 times per minute. He sometimes complained of pain over the præcordial region. The heart was repeatedly and carefully examined, but no endocardial murmur or præcordial friction sound was heard. No thrill or friction-fremitus was felt. He was kept in the recumbent posture, and counter-irritants were applied to the præcordium. At the end of about two weeks he left the hospital, feeling tolerably well, although the cardiac pulsations were rarely below 100 per minute, and a little exercise, or excitement of any kind, would increase them to 120 or more.

In August, 1882, he re-entered the hospital, again suffering from acute bronchitis, with free secretion; numerous sub-crepitant and large moist bronchial rales were detected. In about a week he was convalescent, when a decidedly rough and rather long presystolic murmur was heard. During the remainder of his life he stayed in the hospital, and was engaged most of the time in waiting upon the sick in the ward. Going up or down stairs, or active exercise of any kind, greatly exhausted him, causing marked dyspnœa and tumultuous heart action. He rapidly grew worse, and by the latter part of November he expectorated blood quite frequently. The lungs soon became so engorged, that the frequent hæmoptysis did not relieve them. During most of December he remained in bed, propped up by pillows. The last two weeks the dyspnœa was very marked. His extremities were cool and cyanosed; his face was of a dusky hue, and he expectorated large quantities of blood and frothy mucus. No general dropsy existed. Considerable albumen was found in the urine. Physical signs of pulmonary congestion and œdema, bronchitis, pleurisy with effusion, pleuro-pericarditis, and pericarditis, with effusion into the pericardium, were present during the last few weeks of his life.

Sectio cadaveris.—Numerous recent and old pleuritic adhesions were found, especially in the neighborhood of the heart. The pleuræ were slightly adherent to the upper portion of the pericardium by means of recent exudate. Considerable fluid, containing only a trace of lymph, and no pus, was seen in the pleural sacs. The pericardium contained several ounces of nearly clear serum. Several patches of recently exuded lymph were present on the outer surfaces of the ventricles. The weight and size of the heart were greatly increased. The right cavities of the heart were relaxed and filled with dark fluid blood, and a chicken-fat clot. The left side of the heart was less

relaxed, and contained a smaller quantity of blood. The walls of the right ventricle were nearly twice their usual thickness; its cavity was slightly enlarged. The right auricle was dilated. The valves at the pulmonary orifice appeared competent and show no inflammatory deposits. The tricuspid valves were slightly incompetent, otherwise normal. The left auricle, with its appendix, was enormously dilated. The left ventricle was concentrically hypertrophied. The aortic valves are somewhat thickened, but they were competent, and did not encroach upon the orifice. The mitral leaflets were adherent to each other along their entire right borders, and along the external portion of their left free margins, thus leaving a space only four millimetres long by two wide for the blood to pass through. The valves had a leathery feel, but neither they nor the surfaces of the auricle were rough. The mitral valves do not present the funnel-shaped appearance usually seen in such cases, because, probably, the segments were irregularly adherent to each other, leaving the small opening to one side of the centre of the normal orifice. The lungs were dark, deeply congested, and more or less œdematous. Several ounces of clear serum were found in the peritoneal cavity. The liver, spleen, and kidneys were dark and congested.

One point in the clinical history of this case is worthy of especial attention. The first symptoms, directing attention to cardiac disease, were the rapid pulse and exceedingly irritable condition of the heart. These symptoms existed for several weeks, and probably for a few months before a murmur was audible. An explanation of these symptoms, without the presence of a murmur, will be found by a careful study of the diseased mitral valves before us. Neither the valves nor the surfaces of the auricle are roughened, consequently, for the production of a presystolic murmur under such conditions, it is necessary for the blood-current to meet with sufficient resistance in its passage from the auricle into the ventricle to enable it to set up decided vibration in the valve itself. Before sufficient mechanical obstruction took place at this orifice to develop a murmur, the parts being comparatively smooth, inflammation and beginning adhesion of the leaflets to each other were taking place. The latter conditions, although not sufficient to give rise to a murmur, rendered the heart irritable. If the explanation given is a correct one, it points to the significance of some irritable hearts, where no murmur is present to announce valvular cardiac disease.

The length of the murmur was greater than that of any mitral pre-

systolic murmur that I had heard before. It seemed to be divided into two parts, both occurring between the diastole and systole. The first part was the softer, and had less intensity; the latter was very rough, and ended abruptly. These murmurs corresponded to what Hayden has described as the post-diastolic and presystolic murmurs. He says that they always denote great obstruction at the auriculo-ventricular orifice. The post-diastolic murmur, he thinks, is due to the passive flow of blood from the auricle into the ventricle, the presystolic taking place when the auricle contracts. If subsequent autopsies should almost constantly associate the prolonged or double presystolic murmur with great stenosis at the mitral orifice, it will be of value in prognosis, as life cannot long continue when stenosis is as great as seen in the heart which I exhibit to-night. *May 10th, 1883.*

17. *Mitral stenosis and regurgitation followed by tricuspid regurgitation and general dropsy.*

Presented by Dr. J. T. ESKRIDGE.

Ellen D., 48 years old, single, a servant, was born in Ireland. Her mother died from some chest trouble when about forty years of age. Her maternal relatives were subject to "pleurisies and rheumatism." Her father lived to an advanced age. Ellen enjoyed good health until six years ago, when she suffered from three attacks of rheumatism within a few months. During each attack she was lame in her feet and legs. After these rheumatic seizures, she was comparatively comfortable until the early part of the year 1879, when she noticed that going up and down stairs, or prolonged, or active exercise, exhausted her more than usual, and gave rise to palpitation of the heart. In the year 1880 she had another slight attack of rheumatism. She said her feet were almost constantly swollen during the years 1881 and 1882. Last summer her general health improved, but when the cold weather of the following fall and winter set in, increasing dropsy and dyspnoea returned. She was admitted into the wards of St. Mary's Hospital December 5, 1882, suffering greatly from general cardiac dropsy and associated symptoms. One month later it was noted that she temporarily improved after her admission, but the dropsy recurred, and she failed rapidly.

January 10, 1883, her condition was observed to be as follows: She was jaundiced, irritable, and morose. She dozed frequently, her mind seemed clouded, but she was very restless. The tongue was heavily coated, the breath had an offensive urinous odor, the stomach was irritable, and the anorexia was almost complete. The urine was diminished in quantity, and contained abundance of albumen. Slight effusion existed in the right pleural cavity, but the left was filled up to the lower angle of the scapula. The pericardium contained an increased quantity of liquid. The lungs were congested, and numerous moist bronchial rales were present. Arterial pulsation was seen only in the carotids. Visible venous pulsations were very pronounced in the veins of the neck, and in one or two superficial veins on the anterior surface of the chest. After emptying the veins and exerting pressure upon them, they were observed to fill from the cardiac side, and again pulsate while the finger was still firmly held against the vessels. A wavy impulse, extending over a large area was seen. The cardiac pulsation was most marked just below the lower end of the sternum.

The pulse was very irregular and difficult to count, being about 120 per minute. It was intermittent, and irregular in volume and frequency. The variations of the pulse were most prominent when the hands were raised above the head. The impulse of the heart was felt over a large portion of the anterior surface of the chest. The area of the cardiac pulsation was bounded on the left by a point in the fourth intercostal space external to the left nipple, on the right by a point one inch internal to the right nipple, below by a point two inches below the sternal notch, and above by the left second intercostal space. Hepatic venous pulsation was very distinct. Percussion dulness was increased most on the right side. The pulmonary and aortic valves were apparently free from disease. A presystolic murmur of greatest intensity over the left fourth costo-sternal articulation was heard. A systolic murmur, most distinct over the left fifth costal cartilage, was heard over the anterior surface of the chest from nipple to nipple; also, in the left axilla. It was difficult to determine whether the murmur was audible posteriorly, as the bronchial and crepitant rales and rapid breathing were confusing. Four or five days later the presystolic murmur ceased to be audible. At that time general anasarca was well pronounced. *January 24* she became semi-conscious, which condition gradually deepened into coma. She died *January 26*. She expectorated considerable blood and frothy mucus during the last month of her illness.

Sectio cadaveris twenty-four hours after death.—Body well frozen. Considerable adipose tissue still remained. Thoracic and abdominal cavities only examined.

Thorax.—Left pleural cavity almost completely filled with a thin straw-colored serous fluid. Right pleural sac was nearly half full of a similar effusion. There were no pleural adhesions. The left lung was crowded into a small space and congested; the lower lobe sank in water. The right lung was encroached upon by the effusion, and its lower lobe was consolidated, the upper emphysematous.

The pericardium contained about six ounces of fluid. No adhesions or patches of fibrinous exudations were seen on the surface of the heart. The cavities of the heart were relaxed and filled with dark, fluid blood. The right auricle was greatly dilated. The tricuspid orifice admitted the ends of the thumb and all the fingers of one hand up to the distal joint. The right ventricle was dilated, and its walls thickened. The tricuspid valves were insufficient. The valves at the pulmonary and aortic orifices were thin, but competent. These orifices were not constricted. The walls of the left ventricle seemed thickened, and the ventricle slightly dilated. The left auricle was greatly dilated. The curtains of the mitral valves were adherent to each other near to their attached borders, and constricted the orifice, which they were no longer able to close, into a round opening, only large enough to admit the end of the index finger.

Abdominal cavity.—The bloodvessels of the stomach and bowels were distended. The mucous membrane was softened. The liver was heavy, dark, and grated under the knife. Spleen enlarged, congested, and denser than normal. Pancreas healthy. Both kidneys were congested, slightly cirrhotic, but contained considerable normal tissue.

Remarks.—General dropsy is rare in cases of mitral stenosis, except as in the present instance, where it was combined with mitral insufficiency. No thrill was present during my attendance, which extended over a period of four weeks immediately preceding her death. The mitral presystolic murmur ceased to be audible during the last two weeks of her life. The absence of the presystolic murmur in cases of extreme stenosis of the mitral orifice, late in the disease, when the heart is weak and is acting rapidly and irregularly, has led some observers to believe that the murmur is frequently absent throughout the course of this form of valvular lesion. To this point I directed special attention in a recent paper on the "Diagnosis,

Prognosis, and Treatment of Mitral Stenosis," read at the last meeting of the Pennsylvania State Medical Society. *May 24th, 1883.*

18. *Congenital malformation of the heart, with cyanosis; death at the age of twenty-nine years, from pulmonary tuberculosis.*

Presented by Dr. J. T. ESKRIDGE.

Patrick G., Irish, family history unobtainable, was twenty-nine years old when I first saw him. He had never been strong and able to run and play like other boys, exertion always producing severe palpitation of the heart. So far as he could remember, he did not become blue before his twelfth year. After the occurrence of cyanosis his health became much worse. He had rarely experienced pain in the region of the heart. One year ago he felt sharp lancinating pains through the upper portion of the right side of the chest. These continued and at times were severe. From this time he began to lose flesh and strength, and a dry, hacking cough commenced, but expectoration was not profuse until a few weeks before I first saw him, when he took a heavy cold, which was followed by high fever, great prostration, and profuse sweats. After he had been sick a short time, he was seen once or twice by Dr. O'Hara, who advised his removal to a hospital. He was admitted into the medical ward of St. Mary's Hospital January 2, 1883. Temp. 100°; pulse, 124; resp. 24. He was not greatly emaciated, but was too weak to sit up long in bed. At that time his bowels were constipated, appetite poor, stomach easily nauseated, and he was very nervous. The surface of his body presented a dusky hue, and his face, neck, hands, and feet (especially the fingers and toes), were quite blue. When he sat up the blue color of the mucous surface of the lips deepened into dark purple. The distal phalanges of the fingers and toes were hypertrophied, and the small superficial veins of the face, fingers, and various other portions of the body, were easily seen and counted. Those portions of the body not much discolored by the cyanosis were anæmic in appearance. He was losing flesh rapidly.

Physical signs—Right lung.—The upper portion was consolidated, with signs of a cavity in the apex. The lower lobe was partially infiltrated, but no rales were heard over any part of the lung.

Left lung.—The lower lobe was nearly solid; the upper contained

more air. There were evidences of a cavity. Crepitant and pleuritic friction rales were numerous, especially at the base of the lung.

Heart.—The heart's impulse was wavy, and was seen and felt in the fourth intercostal space, external to the left nipple. A presystolic thrill was felt in the third and fourth intercostal spaces to the left of the sternum, and was barely appreciable in the third intercostal space at the right border of the sternum. The carotid arteries throbbed. I could appreciate no visible venous pulsation. Percussion dulness was increased laterally to the left and right, but did not extend below the fifth costal cartilage. A systolic and a presystolic murmur, with their seats of intensity near the left fourth costo-sternal articulation, were heard. The systolic murmur was audible anteriorly over a large area, and posteriorly at the lower angle of the left scapula, and was heard as low as the seventh rib on each side of the sternum, the sound being faint just below the left clavicle, while it was lost just below the right. It was audible in the left axillary region, but not in the right. The presystolic murmur was not limited to a small area.

January 3, A. M. Temp. 102.3° ; pulse, 120; resp. 30. *P. M.* Temp. 101.5° ; pulse, 118; resp. 28.

4th, A. M. Temp. 101.6° ; pulse, 122; resp. 32. *P. M.* Temp. 102.2° ; pulse, 102; resp. 28.

5th, A. M. Temp. 100.8° ; pulse, 108; resp. 28.

During the above three days he ate well and did not seem to be failing very rapidly, although the cough was troublesome and he was expectorating considerable muco-purulent matter.

6th, A. M. Temp. 101.9° ; pulse, 136; resp. 26. *P. M.* Temp. 101.2° ; pulse, 128; resp. 32. He coughed up during the day several mouthfuls of blood unmixed with mucus.

7th, A. M. Temp. 102.4° ; pulse, 130; resp. 32. *P. M.* Temp. 103.3° ; pulse, 128; resp. 28. There was a profuse expectoration of purulent matter mixed with small quantities of blood. The breath was very offensive. The apex of the right lung was rapidly breaking down. All kinds of mucous rales were heard in the lower portion of the left lung. He was failing rapidly.

8th, A. M. Temp. 101.2° ; pulse, 122; resp. 26. *P. M.* Temp. 103.4° ; pulse, 128; resp. 28. For two days diarrhœa had been very troublesome.

9th, A. M. Temp. 102.6° ; pulse, 106; resp. 30. *P. M.* Temp. 103.2° ; pulse, 116; resp. 28.

10th, A. M. Temp. 101.6° ; pulse, 128; resp. 28. P. M. Temp. 101.4° ; pulse, 128; resp. 32. He had another slight hemorrhage, and diarrhœa was again troublesome, the bowels having been evacuated six times during the night.

11th, A. M. Temp. 101.5° ; pulse, 114; resp. 28. P. M. Temp. 101.2° ; pulse, 116; resp. 30. The cyanotic hue was being replaced by an anæmic appearance. The lungs were breaking down, and considerable effusion had taken place into the right pleural sac.

12th. At 3.30 A. M. he drank about two ounces of wine, and was feeling tolerably comfortable. About half an hour later, when the nurse again visited him, he was dead.

Autopsy.—Body was greatly emaciated, most of the subcutaneous fat having been absorbed.

Thorax.—General pleuritic adhesions were found over the entire left lung, and recent and old adhesions over the upper portion of the right lung. The left pleural cavity was obliterated by adhesions; the right contained forty or fifty ounces of bloody serum. The right lung was compressed and contracted to about half its normal size. Portions of its external surface were covered with pus, tubercle, and bands of adhesion. The lower lobe was consolidated by tubercular infiltration. In the apex was a large cavity, with firm fibrinous walls. The cavity was entirely empty. The upper half of the lung was broken down, and contained numerous small cavities communicating with each other, and with the bronchi. The bronchial tubes and small cavities were nearly filled with pus and frothy mucus. The entire lower lobe of the left lung was infiltrated with tubercle and breaking down. The upper lobe contained numerous nodules and small cavities. The latter, with the bronchial tubes, were nearly filled with muco-pus.

Heart.—Numerous pleuro-pericardial adhesions were present. The pericardium was not inflamed on its internal surface, and was nowhere adherent to the heart, but it contained about two ounces of a straw-colored serous fluid. The heart was anæmic, flabby, and dilated. Its cavities contained a small quantity of fluid blood. No appearances of a heart-clot were seen. The right auricle and its appendix were enormously dilated. The right auricular wall was somewhat thickened. Across this auricle a thin membranous strip stretched from right to left, and from above downward. Its attachment above was at the upper portion of the auricle to the right of the appendix below, at the left margin of the tricuspid orifice. This

imperfect septum seemed to have been an attempt by nature to divide the auricle into two nearly equal compartments. The septum between the auricles was imperfect, the foramen ovale being sufficiently patulous to admit the passage of my thumb from the right auricle into the left. The right auriculo-ventricular orifice was enlarged and admitted the ends of the thumb and all the fingers of one hand. The right ventricle was dilated to nearly twice its normal size. Its wall was not much thickened. A patch of fibroid induration, one inch long by half an inch wide, was seen on its endocardial surface. The anterior and posterior segments of the tricuspid valves formed one large leaflet. The left segment is so situated that it could not have aided materially in closing the auriculo-ventricular orifice. The greater portion of this imperfect segment was stretched across the ventricle near the apex of the heart, more or less obstructing the current of blood from the ventricle into the pulmonary artery. Free regurgitation at the tricuspid orifice was permitted on account of the large size of the orifice, and the imperfect condition of the valves. The other valves of the heart were thin, but competent. None of the cardiac orifices were constricted. The left cavities of the heart were rather small. The pulmonary artery was abnormally small.

Abdomen.—The liver was enlarged, fatty, and grated under the knife. The kidneys, normal in size, were more or less bloodless. The spleen was not enlarged, but it was lying against the cardiac end of the stomach, and firmly adherent to it. No ulcers were found in the bowels.

The slip running across the right auricle of the heart that I present to-night was undoubtedly capable of being set in vibration by the auricular current of blood. If a murmur had been produced thus in the auricle, it would have been presystolic in time. If the blood struggling through the patulous foramen ovale had given rise to a murmur, it would also have been presystolic. The presystolic murmur in this case was well defined, and easily distinguished from the systolic one. It is unfortunate for the science of physical diagnosis that both these lesions existed in the same heart. I know of no way of telling to which condition the presystolic murmur was due. So far as I am aware, a presystolic murmur has never been diagnosed and proved by post-mortem examination to have been due to a current of blood passing through a patulous foramen ovale. In Dr. Mayne's case, in a woman aged thirty-seven years, the deficiency of the inter-

auricular septum was as great as the one which I show to-night, but the only murmur diagnosed by him was systolic in time.

In connection with the description of a case of congenital cyanosis in a boy eleven years old, which I exhibited before the Philadelphia County Medical Society in June, 1882, I quoted, from Walshe, Markham's account of a systolic murmur supposed to have been due to blood passing through an open foramen ovale. "Dr. Fox thinks the murmur ought to have been presystolic, and Dr. Walshe, agreeing with Fox, suggests that it might have been presystolic, and yet mistaken for a systolic one."¹

May 24th, 1883.

19. *Intra-thoracic aneurism.*

Presented by Dr. F. P. HENRY.

This specimen of intra-thoracic aneurism was markedly sacculated, involving the arch and descending portion of the aorta as far downward as the lower border of the sixth rib. The following notes were taken by Dr. Howard Kelly, the then resident physician, soon after the patient's admission to the Episcopal Hospital.

December 7, 1882. There is a "distinctly elevated area about two inches in diameter to the left of the manubrium, occupying the first and second intercostal spaces, and projecting the cartilage of the second rib. This is also the seat of greatest dulness, and of strong bruit and expansile movement. Murmurs are heard at the apex, ensiform cartilage, and second right costal cartilage. Aortic sounds are weak and muffled. Pulse in the right radial and axillary is strong; in left radial and axillary weak, and distinctly delayed. Brachials at elbow are visible, sinuous, and atheromatous. Faint bruit in left axillary; none in right. Strong bruit in left carotid, faint in right. Same relation between subclavian arteries. Faint bruit in the abdominal aorta. Left pupil always smaller than the right."

Under the use of large doses of potassium iodide there was a decided subsidence of the external tumor; also of the pulsation and bruit. The most troublesome symptom, dyspnoea, was not, however, materially benefited.

Death occurred on May 13, through rupture into the right bronchus, and was immediate.

¹ Proceedings of the Phila. County Medical Society, vol. iv., 1881-1882.

The removal of the aneurism was rendered difficult by the adhesions to neighboring tissues, especially to the sternum and ribs in front. The fifth and sixth dorsal vertebræ were deeply eroded, and at the site of these erosions the aneurismal wall was entirely gone, its place being supplied by two masses of fibrin accurately fitting into the erosions, but unconnected organically with the sac. They merely acted as plugs. The heart was in an advanced state of fatty degeneration, and slightly enlarged. The aortic valves were perfectly healthy; but immediately above them were marked atheromatous changes. The opening into the right bronchus was about the *size of a three-cent piece*.
May 24th, 1883.

20. *Specimens from a case of acute phosphorus poisoning.*

Presented by Dr. F. P. HENRY.

These specimens consisted of the stomach, liver, kidneys, heart, and spleen. The phosphorus was taken with suicidal intent during the night of May 7, and was obtained by soaking the heads of a box of matches in water. Fifteen minutes after swallowing the solution, the patient, a male German, æt. 22, experienced a burning sensation in the stomach, which, in the course of a few hours (about six), steadily increased until the pain became excruciating. Copious and repeated attacks of vomiting then ensued, and followed every attempt to allay thirst, which was excessive. On the 8th, there was a very loose discharge from the bowels. The patient was admitted to the hospital on the 10th. His skin was sallow and dark, but not then icteric in hue. There was tenderness over the liver, and the line of liver dulness was slightly increased. Severe pain in the abdomen was complained of, and this pain had continued, with occasional intermissions, since the 8th. The pulse was full and strong; 84 per minute. The temperature was 100°. The urine contained considerable albumen, but no casts, or other abnormal ingredients. I extract the following notes, taken by the resident physician, Dr. James S. Carpenter:—

May 11. No pain. Pulse 100; temp. 98°. Patient refuses food, but craves acid drinks.

12th. Vomited once, but phosphorus not tasted by the patient as heretofore. Pulse 108, and weaker; temp., which was 101° on the evening of the eleventh, now 98.6°. Decided icterus. Thirst continues.

13th. Jaundice increased. Tongue dry and brown, red at edges. Abdominal wall covered with numerous petechial spots. Pulse 126; temp. 100°.

14th. Intense jaundice; one clay-colored stool. Pulse very weak, 122; temp. 98.5°. Extremities cold. Bladder relieved by catheter; 48 ounces removed. The urine contained bile-pigment in large amount, and had a strong odor of phosphorus. The man died at 11.40 A. M., one week, less fourteen hours, after taking the poison.

As the patient was not admitted until the third day after he had swallowed the phosphorus, the treatment was directed toward relieving pain and maintaining the strength as far as possible.

At the autopsy, which was made very soon after death, the stomach was found filled with a grumous, bloody fluid, but the gastric mucous membrane was quite pale, and free from erosion, or any sign of inflammation. The folds of mucous membrane upon its surface were, however, unusually prominent. The liver weighed 3 pounds 14 ounces. The anterior border of the right lobe and the parts adjacent to the gall-bladder were yellow-mottled. Streaks of this yellow coloration extended along the borders of the fissures on the under surface; in parts these streaks were an inch in width. The bulk of the liver was normal in appearance. The gall-bladder was *empty*. Heart rigid in systole; its valves healthy; slight pericardial effusion. Some old pleuritic adhesions and emphysematous vesicles at both apices. Spleen and kidneys apparently healthy. The blood was *fluid*. May 24th, 1883.

21. *Hæmatoma in the upper portion of the mediastinum producing death by pressure upon the trachea.*

Presented by Dr. J. T. ESKRIDGE.

George Boyd, colored, male, aged thirty-two years, porter by occupation, had lived an irregular life. It was not ascertained whether he had suffered from venereal sores. He was slender, and weighed about one hundred and thirty pounds. During the past winter he had suffered from a severe cold on the chest, and had been told by his physician that his heart was affected by the inflammation. With the exception of a slight cough, unattended by shortness of breath, he had considered himself in excellent health until June 10, 1883, when, after carrying a heavy trunk on his shoulder from the first to the fourth floor of a hotel

at Cape May, he was seized with great difficulty in breathing, and had to hold his head and upper portion of his body out of the window and gasp for breath to prevent suffocation. After descending the stairs to the first floor of the hotel, he was again attacked with a paroxysm of asthmatic-like breathing, compelling him to remain in the street until it passed off. He remained in Cape May two days after the occurrence of the accident, and suffered from several paroxysms of shortness of breath during that time. The seizures usually lasted from a few minutes to an hour or more.

He was admitted into the medical wards of the hospital of the Jefferson Medical College on the evening of June 12, about sixty hours after the beginning of his trouble. On admission, his breathing was so short that he could not speak. He was gasping for breath and bathed in profuse cold perspiration. His hands and feet were cool, and nails blue. Respiration 36 per minute, with greatly prolonged expiration; pulse, 100; temperature, 99.7°. He was allowed to inhale amyl nitrite in five- to ten-drop doses until he had used about one drachm. He was also given one teaspoonful of brandy and one-third of a grain of morphia hypodermically. The attack lasted twenty-five minutes. As soon as he was able to swallow, he was given two teaspoonfuls of compound spirits of ether, which was followed by a purgative and a diuretic. He slept well during the night. At 2 P. M. the next day another paroxysm began, but it was promptly relieved by inhalations of amyl nitrite, morphia administered hypodermically, and dry-cupping the chest. He was ordered a pill containing sulphate of morphia, calomel, and tartrate of antimony and potassium, of each one-sixth of a grain, to be given every two hours.

June 14. I saw him for the first time. He was breathing quietly and said that he felt very well. Pulse and temperature were about normal. Urine contained neither albumen nor sugar. No cardiac murmur was detected. Lungs were hyper-resonant over their apices. Loud, moist bronchial rales were heard throughout the lungs, both anteriorly and posteriorly. Remembering his former freedom from seizures of shortness of breath, nothing was detected which was thought to be capable of giving rise to such severe attacks of asthmatic breathing. The development of acute lobular emphysema from over exertion was only surmised, because nothing positive could be found. Potassium iodide and compound spirits of ether were added to the regular treatment. At 5 P. M. he had another paroxysm.

The next two days his breathing was as good as could be expected

with the bronchial trouble present; there were no paroxysms of dyspnoea, and he seemed to be improving. Dry cups were applied to the chest once or twice daily.

17th. During the afternoon he suffered from a severe attack of dyspnoic breathing, lasting several hours, notwithstanding amyl nitrite and morphia were freely used. It seemed to be impossible to nauseate him with tartrate of antimony and lobelia.

18th. At 6 A. M. his breathing became more labored than it had been at any time before, and continued so for six hours. After failing to relieve him with morphia, amyl nitrite, and nauseants, chloral hydrate was employed in fifteen-grain doses, with an ounce of brandy, every hour. This gave more relief than any other agent. Only two or three doses were required before its use was discontinued. During the attack he was delirious a portion of the time, apparently from the intense venous congestion and imperfect oxygenation of the blood. After the severity of the paroxysm was over, his breathing did not become quiet, but remained more or less labored. The night was spent in gasping for breath, restlessness, and moaning.

19th. He was greatly exhausted, partially conscious, and panting for breath. He died quietly at 7 P. M., while fanning himself.

At no time during his illness was pain complained of, except on one or two occasions, when he said his head ached.

Sectio cadaveris was made about twelve hours after death by Dr. Parrott, the medical resident, who has kindly furnished me with an account of the autopsy, and a brief history of the patient's condition while in the hospital.

Thorax.—Pericardial sac was completely obliterated by old firm adhesions of the pericardium to the heart. The heart was rather soft, and both ventricles were relaxed, and contained fluid blood. The mitral and aortic valves were congested and slightly thickened, but all the valves of the heart were competent, and none of its orifices were constricted. Both pleuræ were adherent to the upper portion of the pericardium, and the left pleura was everywhere firmly attached to the lung. No effusion was found in the right pleural sac.

Both lungs were emphysematous at their apices, and considerable lobular emphysema existed. Bronchial tubes were congested, and contained considerable mucus. Bronchial glands were enlarged.

A semi-solid, or rather soft, oblong body, about two inches long by one and a half wide, was found lying upon the lower anterior surface of the trachea, imbedded in connective tissue. It occupied a position

just above and behind the transverse portion of the arch of the aorta, and extended from a point half an inch above the upper surface of the thoracic aorta's transverse arch downward. Three of the rings of the trachea were of a dark color, and one of the spaces between the discolored tracheal rings was about ulcerated through from the effects of pressure. On cutting into the tumor, it was found to consist of partially clotted blood, extravasated into the connective and cellular tissue anterior to the trachea. The extravasated blood compressed the trachea, and greatly lessened its calibre. The bloodvessel that had ruptured was not found. No disease was observed in any of the arteries. No special trouble was seen in any of the abdominal organs.

Numerous cases of rupture of the aorta, or of the smaller vessels, into the trachea, bronchi, œsophagus, or mediastinum, are on record, but in all of these, as far as my knowledge goes, death resulted directly from the loss of blood. The peculiarity of the case I have just described is the formation of a hæmatoma or bloody tumor in the mediastinum. In hemorrhages into this space, the blood usually gravitates to the lower portion of the chest, and the patient soon dies from its effects. In the present case, on account of extensive old and firm adhesions of the pleuræ, pericardium, connective tissue, and everything else in the upper portion of the mediastinum, a hemorrhage in this situation must necessarily have been circumscribed, and could have taken place only gradually by dissecting up the adhesions. The condition of the parts which prevented an extensive hemorrhage, undoubtedly predisposed the bloodvessels, especially the veins, in this locality to rupture. The thoracic aorta, throughout almost the entire extent of its arch, was firmly bound down by adhesions. In view of the extensive alterations that had taken place at the seat of hemorrhage, it is not surprising that rupture of a bloodvessel should have occurred, when the parts in the anterior region of the neck and upper portion of the chest were suddenly put upon the stretch as occurred in the act of raising a heavy trunk from the floor, and placing it upon the shoulder.

From the specimen, as I obtained it, I am unable to say whether the hemorrhage had occurred from a rupture of the aorta, or one of the other large vessels in this locality, or from the bursting of a small bloodvessel which had probably become aneurismal. Tearing across small veins would have given rise to it.

June 28th, 1883.

IV. THE ORGANS OF RESPIRATION.

1. *Some remarks on the pathology of intra-nasal hypertrophies.*

Read by Dr. CARL SEILER.

In spite of the common occurrence of nasal diseases, very little is known about the pathological conditions giving rise to them. The cause of this want of knowledge must be sought in the fact that all portions of the nasal cavities cannot be explored in the living subject, and that, nasal diseases being but rarely fatal, this portion of the body is not, as a rule, included in post-mortem examinations made with a view to determine the cause of death in other diseases; and even in those cases in which it would have been practicable to disfigure the face of the subject by an exploration of the nose, very few investigators have taken the trouble and time to do so. It is true that since the introduction and perfection of the rhinoscope, as well as of the improved methods of inspecting the nasal cavities from in front, much has been discovered which goes to explain the symptoms we notice in nasal diseases; yet there is still a large field left unexplored; and to take a step or two upon the broad expanse of this *terra incognita* is the object of these remarks.

Before, however, entering upon the consideration of the pathological conditions, allow me to say a few words about the anatomy of the nasal cavities and the histology of their lining mucous membrane.

The nasal cavities, which are wedge-shaped, with a narrow arched roof, extend from the nostrils to the upper portion of the vault of the pharynx. Their outer walls are formed by the nasal processes of the superior maxillary and lachrymal bones in front, in the middle by the ethmoid and inner surface of the superior maxillary bones, behind by the vertical plates of the palate bones and the internal pterygoid processes of the sphenoid and the turbinated bones. These latter run from before backward, three on each side, and are designated as the inferior, middle, and superior, the latter being the smallest of the three. The spaces or sinuses between these turbinated bones are called mea-

tuses: so that the space between the floor of the nose and the lower turbinated bone is called the inferior meatus, the one between the lower and middle turbinated bone is the middle meatus, and the one between the middle and superior turbinated bones is the superior meatus.

The nasal cavities are separated from each other by a septum or division-wall, composed of the perpendicular plate of the ethmoid bone and the vomer posteriorly and the cartilaginous septum anteriorly, thus presenting a smooth surface as the inner wall of each cavity.

The floor is formed by the palatine processes of the superior maxillary and palate bones, and runs in a slanting downward direction from before backward. The roof is formed by the nasal bones and nasal spine of the frontal in front, in the middle by the cribriform plate of the ethmoid, and posteriorly by the under surface of the body of the sphenoid bone. Directly communicating with the nasal cavities by narrow channels are other cavities, situated in the bones of the skull, the lining mucous membrane of which no doubt is largely affected by the pathological processes in nasal diseases; these are the antra of Highmore—large triangular cavities situated in the body of the superior maxillary bones and communicating with the nasal cavities by an irregularly-shaped opening in the middle meatus; then the frontal sinuses—two irregular cavities situated between the two tables of the frontal bone. The communication between them and the nasal cavities is established by the infundibulum—a round opening in the middle meatus—and finally the sphenoidal cells or sinuses found in the body of the sphenoid bone, communicating with the nasal cavities by small openings in each superior meatus.

That portion of the nasal cavities which projects beyond the ends of the nasal bones is surrounded by cartilages forming the alæ of the nose.

Malformations in the bony walls of the nasal cavities are by no means rare, and the most common of them is deviation of the septum. This is so frequent that Semeleder found the septum straight in only ten out of forty-nine skulls examined, and Allen¹ found the nasal chambers normal in eighteen out of fifty-eight adult skulls examined. This deviation of the septum must in a great measure be attributed to the fact that at birth both the vertical plate of the ethmoid bone and the cribriform plate are not as yet ossified, and do not become rigid until a much later period of life, and may therefore be easily distorted

¹ American Journal of the Medical Sciences, January, 1880, p. 70.

by external violence applied to the nose by blows or falls. The act of blowing and wiping the nose with the handkerchief must also be considered as a factor in the production of deviation of the septum.

In the cartilaginous septum of the lower animals we find a small cavity lined with mucous membrane, called, after its discoverer, Jacobson's organ, the minute anatomy of which has lately been described by Kline.¹ This organ in man is, however, only rudimentary.

The nasal cavities are lined with mucous membrane, which varies greatly in thickness in different localities, and which materially decreases the size of the cavities in the living subject from that seen in the denuded skull. This mucous membrane is covered by ciliated epithelium in man, with the exception of that portion which lines the vestibule,—i. e., that portion of the cavities of the nose surrounded by cartilage only,—which is covered by pavement epithelium. In the lower animals we find that in the olfactory region the ciliated epithelium is either absent or that ciliated and non-ciliated epithelium alternate in patches.² I have not been able to find a statement in the literature of the subject as to the kind of epithelium found in the accessory cavities in man; but it is very probable that the mucous membrane of the frontal sinuses and the antra of Highmore is covered with ciliated epithelium: otherwise it would be difficult, if not impossible, for the secretions of that mucous membrane to pass through the narrow channels into the nasal cavities. The color of the normal nasal mucous membrane is of a light pink shade in what is termed the respiratory portion, while it is of a yellowish hue in the olfactory region, and in that portion which covers the roof and outer wall of the nasal cavities down to the upper margin of the middle turbinated bone and the septum down to about the same level. It is in this region that the nerve-ends of the olfactory nerve are distributed. Immediately beneath the mucous membrane and between it and the periosteum of the bony walls and the perichondrium of the cartilaginous portion of the septum we find a tissue which bears a striking resemblance to the erectile tissue of the genital organs.³ It is composed of a network of fibrous tissue, the trabeculæ of which contain a few organic muscular fibres. Its meshes, of various sizes and shapes, are occupied by venous sinuses lined with endothelium. These are supplied with blood by small arterioles and capillaries, which are

¹ Quarterly Journal of Microscopical Science, January, 1881.

² Henle, Anatomie des Menschen, vol. ii.

³ Henle, op. cit.

quite numerous in the fibrous tissue, and can readily be demonstrated under the microscope. In this arrangement of elements of the nasal mucous membrane we find a ready explanation of the fact that liquids of greater or less density than the serum of the blood, when introduced into the nasal cavities, produce pain;¹ for we have here the most favorable conditions for osmosis, which will cause either a contraction or a distension of the sinuses. In the larger masses of fibrous tissue between the sinuses or caverns we find embedded the glands, with their ducts opening out between the epithelial cells of the mucous membrane. There are two kinds of glands in this region, which have been described by Kline,²—viz., serous and mucous glands.

This cavernous erectile tissue is most abundant at the lower portion of the septum and the lower turbinated bone; and, although it has been recognized and described as true erectile tissue by Henle, Virchow, and others, yet to Prof. Bigelow, of Boston, belongs the honor of having first called attention to the part which this tissue plays in nasal diseases. He gave to it the name "turbinate corpora cavernosa."³

This short sketch of the anatomy of the nasal cavities will, I trust, be sufficient to enable me to make myself clearly understood when describing the morbid processes and pathological conditions underlying the formation of intra-nasal hypertrophies.

If we closely observe the course of a case of simple acute coryza, we will find that the first symptom is a feeling of fulness, accompanied by sneezing, and that this usually occurs in one nostril at first, the other one being affected later in the same manner. An inspection of the mucous membrane shows it to be in a state of congestion, and so much swollen in certain portions, especially on the inferior turbinated bone, as to touch that of the septum. This produces partial stenosis of the nasal cavity, and is felt as fulness. The congestion having continued for some time, a watery discharge makes its appearance, which is produced by a hyper-stimulation of the serous glands. According to Cornil and Ranvier, lymph-corpuscles are found in this watery discharge of the early stage of acute coryza. Later the discharge becomes thicker by the admixture of the secretion of the mucous glands and of epithelial cells which have undergone fatty degeneration and are thrown off by the rapid formation of new cells

¹ Seiler, Handbook of Diseases of the Throat and Nasal Cavities, p. 97.

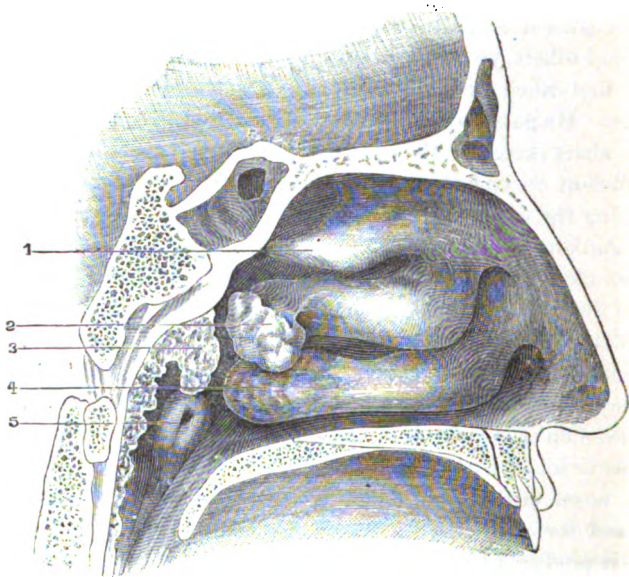
² Loc. cit.

³ Boston Medical and Surgical Journal, April 23, 1875.

under the stimulus of the increased blood-supply. The mucous membrane, as well as the submucous and cavernous connective tissue, becomes infiltrated with numerous leucocytes, and the venous sinuses become distended.

As the acute inflammation subsides, these conditions gradually disappear, leaving, however, the stretched mucous membrane thrown into folds as it contracts, which are especially noticeable at the posterior extremity of the inferior turbinated bone. While spreading, the inflammation involves the glandular tissue situated in the vault of the pharynx, the so-called adenoid tissue or pharyngeal tonsil,¹ and excites it to hypersecretion of the thick yellowish mucus which is expectorated

Fig. 1.



Section of head, showing position of posterior hypertrophy on middle turbinated bone.

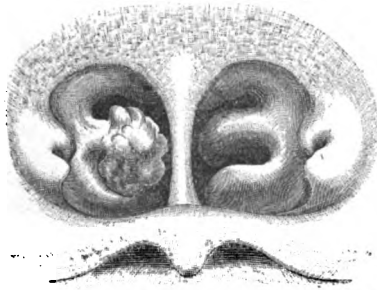
towards the end of the attack. The mucous membrane lining the accessory cavities also participates in the general inflammation, and the accumulation of secretion within them, produced by the obstruction of the narrow outlets by tumefaction of the cavernous tissue, causes the dull pain in the head which accompanies an attack of this kind.

Frequent repetitions of acute coryza at short intervals must of ne-

¹ Luschka, *Der Schlundkopf des Menschen*.

cessity produce a permanency of the inflammatory infiltration in the mucous membrane and submucous tissue, which infiltration finally becomes organized so as to form connective tissue; at the same time the venous sinuses remain more or less distended, and the epithelium of the gland-ducts begins to proliferate. In this way permanent swellings of the mucous membrane in the nasal cavities are formed at the most pendent portions—viz., the lower edge of the inferior and sometimes of the middle turbinated bones; but they are also found on the septum. These swellings are called hypertrophies, and are divided, according to their location, into anterior and posterior.¹ The anterior hypertrophies—those which are situated on the anterior extremity of the turbinated bones or on the cartilaginous septum—are

Fig. 2.



Sketch of rhinoscopic view, showing a posterior hypertrophy in both posterior naris projecting into the vault of the pharynx.

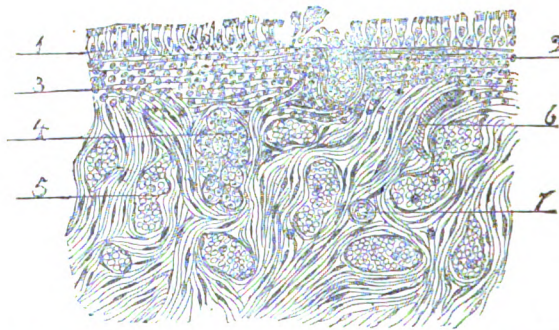
usually sessile and of a bright-red color, while the posterior ones—occurring on the posterior extremity of the turbinated bones—usually have a short pedicle-like attachment and project into the vault of the pharynx. Their color is either a dark-brownish purple or a light-yellowish pink; and I find that those of a dark color are much softer than the light ones. Under the microscope such a condition of the tissues in these swellings is noticed as I have already outlined.

Thus we see in a thin section of one of these hypertrophies that the epithelium is intact, although many of the cells, especially in the neighborhood of the openings of the glandular ducts, have undergone fatty degeneration. The basement membrane upon which the cells are mounted appears thickened, and immediately beneath it we find the mucosa densely infiltrated with a small-celled infiltration, so as

¹ W. C. Jarvis, *The Pathology and Surgical Treatment of Nasal Catarrh*: *Archives of Laryngology*, vol. ii. No. 2.

almost entirely to obscure the mucous tissue. The gland-ducts are seen to be filled with proliferated epithelium, as are also the glands themselves. The bands of fibrous tissue forming the caverns in the erectile tissue are much thicker than in the normal structure, and the venous sinuses are large and irregular in outline. Here and there we find the endothelial lining of these caverns proliferating. Scattered through the connective tissue are seen numerous lymph-corpuscles. In some sections made from hypertrophies I have noticed a myxomatous change taking place in the fibrous tissue. There is but a slight difference in structure between the anterior and posterior hypertrophies—viz., that the venous sinuses in the anterior hypertrophies are not as numerous nor as large as in the posterior variety, and that usually the inflammatory infiltration, as well as the newly-formed connective tissue,

Fig. 3.



Section of posterior hypertrophy, $\times 250$. 1, epithelial layer; 2, mucous follicle; 3, submucosa, showing inflammatory infiltration; 4, mucous glands; 5, venous sinuses filled with blood; 6, small branch of arteriole; 7, transverse section of arteriole.

is much more extended: so that we notice the venous sinuses only near the periosteum when situated on the turbinated bones, and close to the perichondrium when the swelling springs from the cartilaginous portion of the septum.

Thierfelder¹ describes and figures the microscopic appearance of a nasal hypertrophy found by accident in a subject dead from mitral insufficiency, and to the heart-lesion he ascribes the formation of the swelling in the nose. There is, however, no doubt that these swellings are of inflammatory origin, and that in Thierfelder's case of nasal hypertrophy it coexisted with, but was not directly caused by, the heart trouble, as he supposes. The erectile character of the tissues composing

¹ Atlas der Pathologischen Histologie, Lief. 1, Tafel 1, Fig. 1.

the hypertrophies causes them to increase in bulk under certain circumstances. Thus, I have noticed that they are larger in women during the menstrual periods, and probably during the first months of pregnancy. Alcoholic stimulants cause them to swell up, as does mental and sexual excitement,—in fact, anything which tends to increase the blood-pressure in the head. In some cases they are larger in damp weather, while the moisture in the atmosphere does not affect them in others. It is probable that in the first instance they have undergone myxomatous degeneration, giving them hygroscopic properties.

The glandular tissue situated in the vault of the pharynx, and known as the adenoid tissue or pharyngeal tonsil, also becomes involved in the general chronic inflammation, and is likely to become permanently hypertrophied. When thus enlarged, this tissue presents a ragged appearance in the rhinoscopic mirror, with rounded eminences projecting into the pharyngeal cavity. The secretion of this gland, when thus hypertrophied, is a thick, glairy mucus, which tightly adheres to the wall of the pharynx. Detached pieces of the tissue, when examined under the microscope, show the glandular elements greatly increased in number, the epithelium in the glands and ducts proliferating, and the scant connective tissue infiltrated with small-celled infiltration. This condition, however, but rarely interferes with the functions of the nasal cavities, except that it imparts to the voice a nasal sound by decreasing the size of the post-nasal cavity, and thus interferes with the normal nasal resonance, as I have pointed out in a paper read before the American Laryngological Association at its annual meeting in 1881.

On the lower portion of the cartilaginous septum we frequently notice protuberances which to the eye closely resemble the sessile hypertrophies of the mucous membrane, but which, when touched with a probe, have a hard, elastic feel, the same as is conveyed to the hand when touching the cartilaginous septum in other apparently normal portions. These are not localized deviations of the septum—for we do not find a corresponding depression on the other side—but they are true hypertrophies of the cartilage, as I had occasion to prove by removing a very large one and submitting it to microscopical examination. Gottstein claims that they are the result of a localized chronic perichondritis,¹ secondary to the chronic inflammation of the nasal mucous membrane; and this seems very plausible to me, for these

¹ Ueber die verschiedenen Formen der Rhinitis und deren Behandlung vermittelt der Tamponade: Berlin. Klin. Wochenschrift, No. 4, 1881.

cartilaginous hypertrophies are met with only in cases of long-standing catarrh.

On the floor of the nose we frequently see bony excrescences springing from the superior maxillary bone, which were described by Dr. Allen.¹ These are usually congenital, and, unless they give rise to pain and inconvenience by pressure through their size, are harmless.

According to Virchow's definition, these hypertrophies should be considered as tumors (which would be a strong point in favor of my friend Dr. Formad's inflammatory theory of tumors); but, inasmuch as they are not true neoplasms, but localized increase of size of the normal tissues, and as they are not permanent—often atrophying without having previously undergone destructive changes—they cannot be considered as such; and the term hypertrophy, which has been used to designate them, is, in my opinion, a proper one. There is, however, a class of tumors, so called, found in the nasal cavities, which, springing from the mucous membrane or periosteum of the turbinated bones, or more rarely from the septum, differ in their histological elements, as well as in shape and size, from the hypertrophies—viz., nasal polyps.

Two varieties of nasal polyps are usually recognized—the mucous and the fibrous variety—to which I would add a third—the cystoid.

Like the hypertrophies of the mucous membrane and of the cartilaginous septum, these polyps are due to inflammation; and Galen recognized this fact, for Virchow² quotes him as saying “that the nasal polyps are due either to inflammation or develop from a node or from germinal matter.” And Virchow himself³ says that on mucous surfaces tumors for the most part occur in places where there previously was a simple inflammatory disturbance—where the simple inflammatory hyperplasia of chronic catarrh precedes the growth of polyps.

It is therefore evident that they may occur on any portion of the nasal mucous membrane, and that they will be found more usually in those portions of the nasal cavities which are most exposed to the irritating influences of the air and dust—viz., in the respiratory portion. They are, however, also found in the antra of Highmore.

Under the microscope the mucous variety is seen to be composed chiefly of myxomatous tissue, which is intermingled with fibrous tissue and some organic muscular fibres. Embedded in their substance we find some hypertrophied glands as well as venous sinuses, and sometimes we find in thin sections openings lined with columnar epithelium, which are probably the cross-sections of invaginated portions of mucous

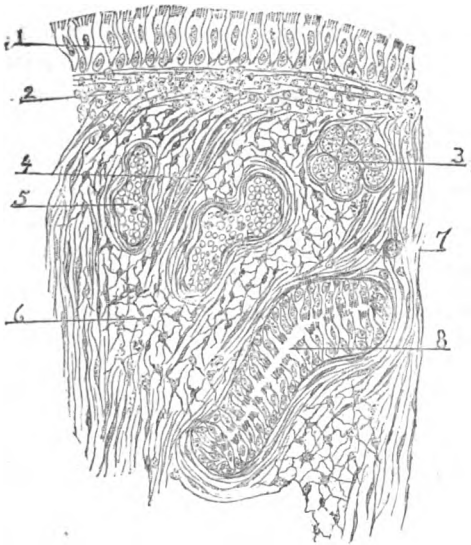
¹ Loc. cit.

² Die Kr. Geschwulste.

³ Loc. cit.

membrane. The polyps are covered with ciliated columnar epithelium in those portions which are not exposed to the direct influence of the air, while the convexities pointing towards the nostrils are covered with stratified epithelium. Billroth¹ describes them as retaining all the elements of the mucous membrane from which they sprang. Occasionally we find that they have undergone teleangiectatic degeneration.

Fig. 4.



Section of mucous polyp, $\times 300$. 1, epithelial layer; 2, infiltrated submucous layer; 3, mucous gland; 4, fibrous band; 5, venous sinus filled with blood; 6, myxomatous tissue; 7, transverse section of arteriole; 8, invagination of mucous membrane.

The more rare fibrous variety, which is very hard and of a glistening white color, stands in contrast to the soft, gelatinous, pinkish, and highly hygrometric mucous variety. Cornil and Ranvier, say of the fibrous polyps, "They usually have their point of attachment in the posterior portion of the nasal cavity. They send prolongations in every direction, into all the cavities, either bending around obstacles or breaking through them, enlarging the nasal fossæ, thinning or destroying the bones, and penetrating by new ways or natural openings into the sinuses which surround the nasal fossæ."

Under the microscope they appear as true fibromata, containing, however, like the mucoid variety, glands, venous sinuses, and nume-

¹ Ueber den Bau der Schleim-Polypen, Berlin, 1855.

rous capillaries. Both the fibrous and the mucoid variety of polyp are not infrequently combined in the same growth.

The question has arisen in my mind whether these growths could not be looked upon as simple hypertrophies of the mucous membrane which have undergone mucoid degeneration or fibrous change, or both, as the case may be; for in this way the presence of glands, venous sinuses, and spaces lined with epithelium within their structure can readily be explained, while, on the other hand, the presence of these foreign elements cannot so easily be accounted for, if we consider the polyps genuine neoplasms. Having once started in a localized hypertrophy of the mucous membrane, the mucoid or fibrous change rapidly assumes large proportions under the stimulus of continued irritation, pushing the mucous membrane before it; and in this way the often enormous pear-shaped masses are produced. I have frequently found a number of small mucoid polyps on the mucous membrane near the site of larger ones which I had previously removed, and which, if left undisturbed, would soon have filled the nasal cavity by their increase in size. This is a question, however, which cannot be determined by merely examining extracted polyps, but may possibly be settled by making sections through the mucous membrane at the point of origin of the tumors; but as yet I have not had either the material or the opportunity to do so.

The third variety of polyp is a large sessile cyst filled with thin watery mucus and covered with epithelium. In the few cases which I have seen—too few to make extended examinations as to the nature of these growths—they sprang from the lower border of the inferior turbinated bone. I have not met with a mention of them in the literature to which I had access.

All these conditions of the nasal cavities produce either partial or complete stenosis, thus interfering with the physiological functions of the nose, the consequences of which, as well as the treatment adapted for their removal, I have pointed out in a paper recently read before the Philadelphia County Medical Society¹ and in an article published in the *Medical Record*.²

If by these remarks I have succeeded in stimulating others to pursue this interesting subject, and in causing an interchange of ideas in the discussion of it, the object of my feeble efforts has been fulfilled.

¹ Surgical Treatment of Nasal Catarrh: *Medical Times*, October 8, 1881.

² Jarvis's Operation, with Report of Three Cases: *Medical Record*, October 29, 1881.

Discussion.—Dr. Cohen in opening the debate, said that he agreed with Dr. Seiler that most polypi were the result of the irritation consequent upon frequent attacks of coryza. As Dr. Seiler had mainly confined his remarks to the results of the microscopic examination of these hypertrophies, he could not criticize them, having made no such investigations himself; but as to the necessity for their surgical removal he would merely say that in his experience any such measures were but rarely called for.

Dr. Nancrede referred to the frequency of deviations of the bony nasal septum, instancing over one hundred skulls, in fully one-half of which number he had observed that the septum was more or less deviated.

Dr. Seiler, in closing, said that he had not referred to the pathology of true polypi, but to what he had termed intra-nasal hypertrophies. He regretted that no one had anything to offer with regard to the *serous* glands mentioned in his paper. To their secretion he attributed the fact that the throat never became dry during sleep, when the individual breathed solely through the nose, but that dryness invariably resulted if the mouth remained open. In both the nose and mouth mucous glands are abundant; but they clearly do not supply enough moisture for respiratory purposes, and in consequence the normal respiratory passages—viz., the nasal—have superadded special glands for moistening the inspired air,—viz., the serous.

November 10th, 1881.

2. *Organs from a case of acute tuberculosis.*

Presented by Dr. H. M. FISHER.

Helen L., æt. 4. Father is a negro, mother a mulatto. Mother appears delicate; but no history of consumption in the family of either parent can be obtained. Child had always been considered healthy, and does not appear to have suffered from any of the usual diseases of childhood. I saw the case first November 12. About six weeks previous to my first visit, the mother noticed that the child was losing her spirits and appetite, was languid and listless, and complained of some abdominal pain. She noticed at the same time that the child's skin was hot and dry. No hæmoptysis occurred, no cough was noticed at this time, and there were no night-sweats. There had been no

vomiting, no pain in the head was complained of, and no convulsions at any time occurred. The child had one small loose passage daily. She slept much during the day, and was rather restless at night. There had been at the same time progressive emaciation.

Upon examination, I found that the child was thin, but not markedly emaciated; the skin was dry, and on the legs and thighs there was considerable epidermic exfoliation. The abdomen was moderately distended with gas. Tongue presented strawberry appearance from enlarged papillæ. Pulse, 120; temperature, 102.2°. Respiration perhaps somewhat quickened, but not notably so, and there seemed to be little or no dyspnœa. I was able to detect no dulness on percussion of the chest, nothing abnormal in the respiratory murmur, and no adventitious sounds. The child was very apathetic, and could not be induced to answer questions; but there was nothing that seemed to point to cerebral lesion.

November 23, eleven days from date of my first visit, I saw the child again. I was informed that for about a week a slight cough had been noticed, and that the emaciation had been rapidly progressing. This was indeed marked. The fever persisted (101.4°). Respiration about 40, but no marked dyspnœa. A careful examination of the lungs revealed some impairment of percussion resonance over the upper lobes of both lungs; but I could not convince myself of any impairment of resonance over the lower two-thirds of the chest.

Prolonged expiration and a few fine rales were heard in the right supra-scapular fossa, but over the rest of the lungs I detected nothing abnormal.

Death occurred three days later, without the manifestation of further symptoms.

Brain not examined.

Post-mortem examination, forty-two hours after death. — Rigor mortis has disappeared. Both lungs appear freely movable, and no adhesions can anywhere be detected. The pleural cavity appears to contain a normal amount of serum. Pericardial cavity contains also a normal amount of light straw-colored serum. Left ventricle dilated, and contains black blood and a soft black clot. The left ventricular wall appears thin, and the entire heart-muscle is flabby. The right ventricle contains also black, partially coagulated blood. The orifices appear to be of normal size, and no valvular lesion can be detected. Bronchial glands enlarged, but not cheesy. The external surface of both lungs presents a general dusky red hue, with some yellowish-

white mottling. This "marbling" of the tissue is most pronounced in the lower third of both lungs. The tissue of both lungs crepitates freely on pressure. The lungs do not float perfectly when placed in water. Upon section, a moderate quantity of frothy muco-pus exudes. The cut surfaces of both lungs present numerous fine grayish white granulations. These appear to be somewhat more numerous in the upper than in the lower portions, and the left lower lobe is tolerably free from them.

Abdominal cavity.—The bladder is found to be largely distended with urine, its upper surface extending fully four inches above the symphysis. The intestines are considerably distended with flatus, but present—at least on their external surface—no evidence of tubercular granulations, and there are no adhesions or other indications of peritonitis. The spleen is much enlarged and of a mahogany-red color. Upon section, its cut surface is found to be granular. With tinct. iodinii the reaction indicative of amyloid degeneration is not produced.

Both kidneys are pale and flabby, and in both the capsule is easily removed without tearing the organ. Upon section, the cortical portion is found to be pale and opaque, swollen and slightly mottled, and in some portions of the section numerous grayish-white nodules are seen, none of which are larger than a pin's head.

The pancreas appears normal. I neglected to open the stomach and intestines, but externally I could see no evidence of inflammatory changes.

The liver is greatly enlarged, its right lobe extending one-half inch to the left of the xiphoid cartilage and about three inches below the ribs, while the left lobe extends fully four inches to the left of the xiphoid cartilage and about two and one-half inches below the lower margin of the ribs on the left side. The tissue of the liver is pale and opaque, and has a doughy feel. A few whitish nodules, similar to those in the kidney, were seen in the liver.

With the advice and assistance of Dr. Longstreth, I made the microscopical examination.

Lungs.—Tubercular aggregations are very numerous, but very few of these present central cheesy degeneration, being pretty uniformly stained from the centre to the periphery. The portions of lung-tissue adjacent to the tubercular granulations show catarrhal inflammatory changes, while other portions of the lung are nearly free from them.

On the other hand, the lung-tissue is seen to be hepatized in many places between nearly adjacent tubercles.

The *liver* presents numerous small tubercular masses in an early stage of formation, and the liver-cells show in places moderately fatty infiltration; but the most marked feature is the general atrophic condition of the cells. The tubercular masses are found chiefly in the interlobular connective tissue. There are but few of these masses within the lobules themselves.

Spleen.—None of the sections show well-defined tubercular nodules; but there are numerous aggregations of brightly stained nuclear bodies scattered through the organ, and the elements of which these are composed entirely resemble the elements of newly-growing tubercle. The Malpighian bodies present no alteration, neither do the fibrous trabeculae, except in portions occupied by the newly-growing tissue.

Kidneys.—In one section of the kidney, from cortex to pyramid, six tubercular nodules were counted. The largest and most fully developed nodules are in the region of the base of the pyramid. None of the tubercular nodules show any limitary membrane, but they pass off gradually into the surrounding tissue. The tubules which pass through the growth are not entirely destroyed; the capillaries, however, are pressed upon and obstructed. There are areas of the cortex where there is infiltration of nuclei. The endothelium of the tubuli is for the most part granular, but not sufficiently so to obscure the nucleus. There are no fatty changes present. The glomeruli and the arterioles are unchanged.

December 8th, 1881.

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3. *Ecchondroma of the larynx; ankylosis of the right arytenoid cartilage; dyspnœa, aphonia; death from pneumonia following tracheotomy.*

Presented by Dr. J. H. MUSSER.

The patient from whom this rare specimen was removed applied to the University Hospital medical clinic for treatment, having been sent by Dr. Wetherill, of Lambertville, New Jersey. Prof. Pepper, in a clinical lecture, developed the following facts of the case.

During the war the patient was a cornet-player, continuing his profession since then, as his health permitted. Ever since the war he complained of irritation in the throat and of shortness of breath, while his voice was changed in tone,—cracked. The dyspnœa was worse on exertion. There was gradual loss in the power of the voice. Three

years ago he had to give up work, especially on account of dyspnœa. His breath had been offensive.

When examined he was 50 years of age. He had lost twenty pounds in weight. The voice was lost; dyspnœa was constant; the breathing was stridulous; deglutition was not difficult, but there was a sense of obstruction; there was no expectoration. Laryngoscopic examination revealed congestion of the parts about the base of the epiglottis, and of the ary-epiglottic folds. There was complete paralysis of the right vocal cord; it was drawn aside; the right arytenoid cartilage did not move. Below the vocal cords, on the posterior wall, a tumor was readily seen, encroaching upon the lumen of the tube, the only free space being to the left of the median line.

The patient was advised of his danger, and of the wisdom of tracheotomy. He went home, to return in a short time on account of urgent dyspnœa. Just after entering, the dyspnœa became so severe that tracheotomy had to be performed hurriedly, May 28, 1881. He rallied well from the operation, and was in a good condition until June 1. Pneumonia developed that day, resulting in death in forty-eight hours.

After death, the state of the larynx was found as noted above. A tumor the size of a walnut, of the macroscopic appearance of an enchondroma, grew from the right half of the posterior surface of the cricoid cartilage into the lumen of the larynx. A space, elliptical in shape, one-half inch in length and one-eighth in width, to the left and anteriorly, alone permitted the entrance of a probe. The arytenoid cartilage was immovable.

It is of interest to note the causal relation between the occupation of the patient and the laryngeal disease, and to consider the inflammatory origin of the mass. In the works which I have at my command I cannot find any records of a similar case. Hence it must be extremely rare.

Dr. J. Solis Cohen said that he had never seen any such growth reaching the large size this one had attained. Such tumors were common in Europe, but much rarer here. *January 26th, 1882.*

4. *Fibroid polypus of the nose involving the antrum.*

Presented by Dr. C. SEILER.

A. L., a farmer, æt. 68, was sent to me by Dr. Woodward, of West Chester, early in December of last year, for operation. I examined the patient, and found the left nostril completely closed by a hard, lobulated mass. The left cheek-bone was very prominent, and the left eye was pushed out of the orbit so as to be exceedingly prominent. In passing my finger over the outer wall of the antrum, I discovered a triangular opening in the bone, through which a soft mass was protruding. The affected eye was constantly weeping, and the patient complained of severe neuralgic pain in the left side of his face. He stated that he had noticed several years back that his nose was stopped up, and the pain in the face had made its appearance about six months since. With the wire snare I removed several pieces of the growth from the nostril, and on examination found them to consist of fibrous tissue. The diagnosis of fibroid polypus involving the antrum was then made, and the patient referred to Dr. J. Ashhurst, Jr., for operation.

A few days later, the patient was operated upon in the clinic at the University Hospital, and this large mass of fibroid polypus was removed. During the operation it was found that the anterior wall of the antrum as well as the orbital plate had been almost entirely absorbed, and the turbinated bones on that side of the nasal cavity had disappeared. The hemorrhage was very profuse, and the patient died an hour after the operation was finished.

Dr. Barton had seen several similar cases, but they had all proved to be malignant. He would therefore suggest the propriety of making sections of the whole growth, when portions might then be seen which would be of a malignant nature.

Dr. Seiler thought that the cases described by Dr. Barton were not true fibromata, but *fibrous polypi*, in which were found invaginated mucous membrane, angiomatous tissue normal to the inferior turbinated bones, etc. Nasal fibromata are usually single, unlike ordinary polypi. The former usually *press* the septum to one side, while they induce *erosion* of the superior maxilla.

Dr. Tyson could corroborate the last portion of Dr. Seiler's remarks from personal observation.

March 9th, 1882.

5. *Tuberculosis as manifested in the larynx.*

Read by Dr. J. SOLIS COHEN.

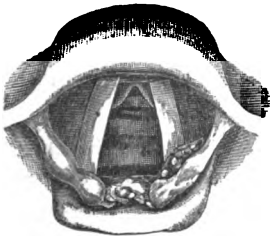
Tuberculosis, as manifested in the larynx, includes, as discussed in this paper, the entire range of pathological changes which ensue in the larynx as a result of its infiltration by tubercle. Attention will be directed both to the clinical pathology of the malady as revealed by laryngoscopy, and to its histological pathology as revealed by microscopy.

Following the resort to laryngoscopy as a means of objective diagnosis, announcements were soon made that the early presence of miliary tubercle could be detected in the mucous membrane of the living larynx; and that the entire progress of the tuberculous process could be studied from time to time in the laryngoscopic image. Similar assertions, indeed, are still made.

Small globular or semiglobular nodules, pin-head or thereabouts in size, yellowish in tint, seen isolated or clustered at different portions of the laryngeal mucous membrane, were attributed to accumulations of miliary tubercle.

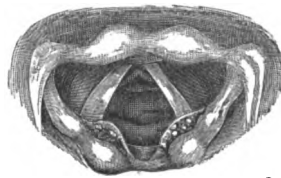
The customary destructive metamorphosis of these bodies, long before the death of the patient, prevents verification or denial of their initial tuberculous character upon positive premises. By prolonged observation, however, it has become demonstrated that these tuberculous-looking nodules (Figs. 1 and 2) always occupy localities normally

Fig. 1.



Distended mucous glands.

Fig. 2.



Distended mucous glands (of three years' standing).

beset with mucous glands;¹ whence the inference has arisen that they

¹ Inner surfaces of the arytenoid and supra-arytenoid cartilages, lower and inner surface of epiglottis, meso-arytenoid fold.

are not tubercles; but are rather hypertrophically distended mucous glands, filled, by occlusion of the orifices of their ducts, with accumulated products of secretion and desquamation; inflamed, perhaps, by some specially irritative quality in the hyper-secretions of the coexisting chronic catarrhal laryngitis. The ultimate destruction of these glands results chiefly from necrotic inflammatory processes set up by the pressure of tuberculous infiltrations around them and between their individual acini. In this manner follicular ulcerations are produced whose racemose configuration so closely resembles the crenated margins of some tuberculous ulcerations, as to render it often impossible to distinguish one from the other, save under the lens of the microscope.

Nodules, similar in their gross laryngoscopic aspect to those which have just been mentioned, sometimes remain unchanged for many months. Several examples have occurred in my own practice; the appearance depicted in the second illustration (Fig. 2) having lasted, to my knowledge, for more than three years, in the larynx of a practising attorney of this city. These certainly cannot be tubercles, except under the questionable hypothesis of their calcification.

The long-mooted question of the existence of tubercle in the larynx, seems to have been set at rest in the affirmative; and chiefly by quite recent researches of Heinze¹ and Eppinger.²

Primary tubercle.—Pathologists acknowledge the possibility of primary infiltration of the larynx with tubercle; but they await satisfactory confirmation of the hypothesis. No record exists, to my knowledge, of detection of tubercle in the larynx of the dead subject, without abundant coexisting tubercle in the lungs. Clinical evidence of such primary deposit is presumptive rather than demonstrative. This presumptive evidence is based solely upon laryngoscopic inspection, which, in individuals in whom no physical signs of pulmonary lesion can be detected, reveals a condition of the larynx known to be more or less characteristic of tuberculous processes in that structure.

Secondary tubercle.—Secondary infiltration of tubercle in the larynx is generally acknowledged to be of comparatively frequent occurrence. It takes place, as a rule, only in the subjects of pulmonary tuberculosis; and, as far as my own records teach, appears much more frequently in the inherited than in the acquired variety. It is, furthermore, asso-

¹ Die Kehlkopfwindsucht, nach Untersuchungen im pathologischen Institute der Universität: Leipzig, 1879.

² Pathologische Anatomie des Larynx und der Trachea: Berlin, 1880.

ciated, as a rule, with secondary tuberculosis in other structures; both at a distance, *i. e.*, intestines, spleen, kidneys, etc.; and contiguous, *i. e.*, trachea, pharynx, palate, tongue, etc.

An *acute tuberculous sore throat* has been described, with considerable detail, by Isambert, Frænkel, and a few others. It is an acute miliary tuberculosis of the pharynx and larynx, which rapidly ulcerates, and terminates fatally in a few weeks, under further progress as acute tuberculosis of the lungs. Abundant disseminations of confluent patches of miliary tubercle have been observed beneath the epithelium, which bleeds freely when touched. These appear first upon the palate, anterior palatine folds, the tonsils, and the pharynx; and, at a later stage, upon the epiglottis and the larynx. They are exceedingly painful, so much so that deglutition is sometimes impracticable. Ulceration soon ensues, enucleating a certain number of the tubercles; and leaving empty sacs, with more or less deep losses of substance. Death occurs, usually, before extensive ravages can be produced.

Of this affection I know almost nothing personally. One example presented at the Throat Clinic of Jefferson Medical College Hospital, a few years ago, in the person of a lad, whom I had but the one opportunity of examining; and who, as I learned upon inquiry, died a few weeks afterwards.

In 1868, the larynx, from what I strongly suspect to have been a case of this kind, was presented to this Society, by Dr. Tyson,¹ who called attention to the fact that the ulceration began in the fauces, and that the patient, a man 49 years of age, whom he had seen in consultation, suffered with painful deglutition to an extreme degree. The rapid progress of the disease in this instance, the intense pain on deglutition, the early ulceration in the throat, and the slight amount of laryngeal ulceration found post-mortem, tally very closely with the pathological history of the cases discriminated of late years as examples of acute tuberculosis of the throat.

Presumptive primary tuberculosis.—My entire practice has furnished me with but three personal examples of even presumptive primary tuberculosis of the larynx. In two instances it was impossible to detect evidences of pneumonic lesions for several weeks following recognition of the tuberculous larynx. The subjects were all males; aged, respectively, 29, 27, and 21 years.

In two cases, one a driver of an ice-wagon, and the other a sailor,

¹ Trans. Path. Soc. Phila., vol. iii. p. 74.

the immediate advent of the lesion was directly attributable to severe cold; probably acute laryngitis, from extreme exposure. The third patient, a miller, had no recollection of having caught cold. Hereditary influence was denied in each case.

In the sailor, pneumonic symptoms first became discernible six weeks after the manifestation of disease in the larynx; and death occurred by apnoea within ten weeks thereafter.

In the driver of the ice-wagon, pneumonic symptoms first became discernible eighteen weeks after the manifestation of disease in the larynx; and death ensued eight weeks later. In the miller, the first pneumonic symptoms became discernible fourteen weeks (April 3, 1882) after the disease had begun; and at last accounts he was reported as far gone in pulmonary tuberculosis.

CASE I.¹ (No. 17,250).—The first laryngeal lesion, recognized, was a shallow irregular ulcer on the left side of the posterior face of the pallid and thickened epiglottis. Ulceration soon attacked the right side also; then the central portion of the edge of the epiglottis, and subsequently its laryngeal face. Thus the epiglottis became encircled, as it were, with an ulcerating girdle, and gradually underwent destructive ulceration from above downward, till nothing but a hemorrhagic stump remained. At the autopsy it was found that the ulceration which had surrounded the epiglottis had extended into the base of the tongue, and had destroyed a portion of its substance. The ulceration on the laryngeal surface of the stump of the epiglottis was quite extensive, as was that also on the aryteno-epiglottic folds and the ventricular bands. The vocal bands were intact, as was also the whole of the subglottic mucous membrane of the larynx and of the trachea, as far as it had been removed. This is distinctly shown in the specimen herewith presented.

Dr. Seiler kindly made a number of sections of this larynx, two of which are now under the microscope for inspection. One shows small-celled infiltration and caseous degeneration in the stump of the epiglottis; and the other exhibits infiltration with cheesy centres in the mucous membrane, and infiltration in a mucous gland.

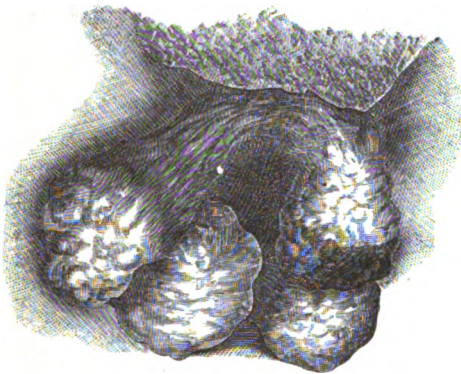
Tubercle was abundant in both lungs in various stages of degeneration. Several small cavities were seen in the upper lobe of the left lung; but there were none in the right lung.

¹ For details with illustrations, see *Archives of Laryngology*, vol. ii. No. 2.

CASE II. (No. 21,110).—Geo. F., aged 29, a German blonde, unmarried, and for fourteen years a seaman, had no record of sickness prior to six weeks before being sent to me for laryngoscopic examination. Exposed to very cold weather in the English Channel, he acquired what was probably an acute laryngitis, attended by dysphonia, dysphagia, cough, and expectoration. The dysphagia increased until swallowing had become exceedingly difficult and exquisitely painful. His pain, indeed, was the principal subject of complaint.

Nutrition seemed good. Lung capacity was of normal average. There was no sign of dyspnoea on exertion. There was slight dulness on percussion at the apex of the right lung; and bilateral exaggerated vocal resonance on auscultation posteriorly. The mucous membrane of the gums of the upper teeth was studded with tuberculous-looking elevations.

Fig. 3.

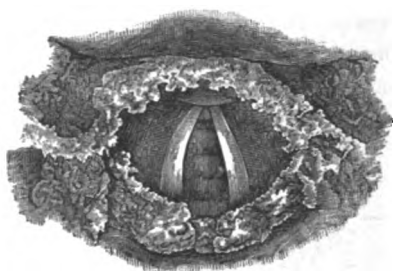


Ulcerative acute tuberculous laryngitis.

Laryngoscopy revealed (Fig. 3) almost complete ulcerative destruction of the right half of the epiglottis. The ulcerations extended into the glosso-epiglottic sinuses in the one direction, and into the aryteno-epiglottic fold in the other; the glosso-epiglottic ligament and aryteno-epiglottic fold being destroyed to a considerable extent. The ulceration extended into the base of the tongue on that side. A few red unhealthy granulations existed at the junction of the crest of the epiglottis with its left side; ulceration existing over the whole of that side of the epiglottis, also, but much less deeply than on the right side. The left edge of the epiglottis was several times the normal thickness;

and a deep oval excavated ulcer occupied its free edge. There was immense tumefaction of both supra-arytenoid eminences. The left side of the larynx was completely hidden; and the interior of the right side indistinguishable. Progressive ulceration gradually destroyed so much of the swollen epiglottis and aryteno-epiglottic folds as to fully expose the interior of the larynx to inspection (Fig. 4), when it was seen that the vocal bands were intact, as had been inferred from the character of the voice.

Fig. 4.



Progressive ulceration in acute tuberculous laryngitis.

Post-mortem examination revealed complete tuberculous infiltration of the right lung, and almost equally extensive infiltration in the left lung; only a few cubic inches in the anterior portion of the lower lobe being free from the product. The lungs contained no vomicae.

There was extensive ulceration of the base of the tongue, the remnant of the epiglottis, both aryteno-epiglottic folds, and both lateral laryngeal walls almost to the edge of the ventricular bands. No macroscopic lesions were apparent on the vocal bands, or in the subglottic portion of the larynx, or in the entire trachea, or in so much of the primitive bronchi as were removed with the specimen, which is herewith presented for inspection.

In both these cases the tuberculous lesions are limited to the supraglottic portion of the larynx, as was likewise the case at the last laryngoscopic examination of Case III., the pathological particulars of which I shall, probably, be prepared to present to the Pathological Society at no distant date.

As to the etiology of these cases, we are restricted to hypotheses. There is no positive evidence of hereditation. In two, there was distinct origin in a severe cold, most probably an acute laryngitis.

In the third, there was no recollection of any special cold; but it is

not improbable that the disease began as an acute or subacute laryngitis, or laryngo-bronchitis, milder in character than in the other cases. It is quite possible, further, that there may have been some slight pneumonitis accompanying the laryngitis or laryngo-bronchitis in these cases, and that the caseous foci of some of its residual products in the lung originated the tuberculization.

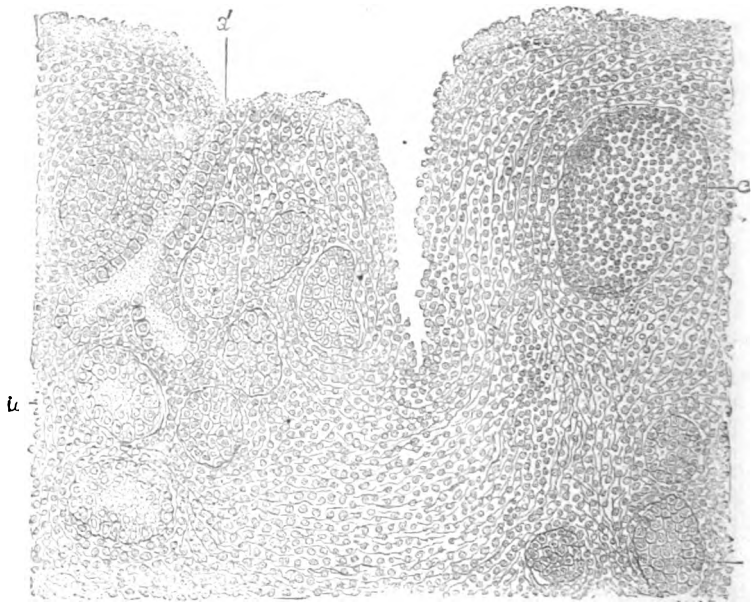
On the whole, therefore, I am inclined to the belief that cases of so-called primary tuberculosis of the larynx may be relegated to the category of secondary tuberculosis, commencing very early and running an unusually acute course.

Secondary tuberculosis of the mucous membrane of the larynx presents us with two stages: (1) that of infiltration; and (2) that of ulceration; several specimens of both of which conditions are under the microscopes before you. No tubercle is found in the epithelium; the infiltration always taking place beneath the epithelium. The infiltration is found both in the mucosa and the submucosa; in the latter, however, rarely as deeply as the situation of the mucous glands; according to some, never; but this negation is too absolute, as will be proved by several sections now under the microscope (Fig. 5). One section, through a ventricular band, exhibits granulation tubercle extending more and more densely in the very vicinity of the glands, everywhere infiltrating the interacinal connective tissue, and in many places so profusely infiltrating the glands as to render it difficult to tell whether a given mass is a tubercle or an infiltrated gland. In some instances, as in some of the specimens before you, the infiltrate is uniformly disseminated through the entire thickness of the mucous membrane; but in the great majority it is found only in the upper part of the mucosa, just beneath the epithelium. Sometimes there is quite a free space (Heinze) between the epithelium and the most superficial tubercles. The overlying epithelium appears normal; and remains well attached unless ulceration has actually begun. Individual tubercles are noticed more abundantly in the upper portions of the mucosa, and more and more sparsely towards the deeper. In these portions, too, there is less granular infiltration than there is above. In the sections exhibited, the older tubercles occupy the central portion of the mucous membrane chiefly; young ones, the subepithelial portion. Giant cells are few in number. Advanced cases exhibit extensive caseation, both in the tubercles and in the tissue immediately contiguous; especially near the periphery.

Miliary tubercle is beautifully exemplified in one of the preparations

under the microscope, from the larynx of an infant seven months of age. The section, for which I am gratefully indebted to our accomplished curator, Dr. Seiler, includes the entire circumference of the larynx directly through the glottis, and the tubercles in the field of the instrument are located in the inter-arytenoid fold.

Fig. 5.



Tuberculous ulceration; involvement of gland. Section through mucous membrane at base of epiglottis; *aa*, gland and its duct infiltrated with granular tubercle; *b*, acinus of gland; *c*, tubercle. $\times 1:0$.

Circular infiltration occurs partly outside the adventitia of the blood-vessels; but also, and to a greater extent, imbedded between its fibres. Fully formed tubercles are sometimes observed; occasionally with evidence of central caseation. The lumen of the vessels is obliterated by pressure in many places. Extensive infiltration has destroyed parts of the adventitia; but the integrity of the remaining coats of the arterial vessels is usually well maintained. So likewise with the capillaries; while the more delicate tunics of the veins readily undergo destruction; mere traces remaining in some localities.

As regards the glands, to ulceration of which a tuberculous character has been so much attributed both by many clinicians and not a few

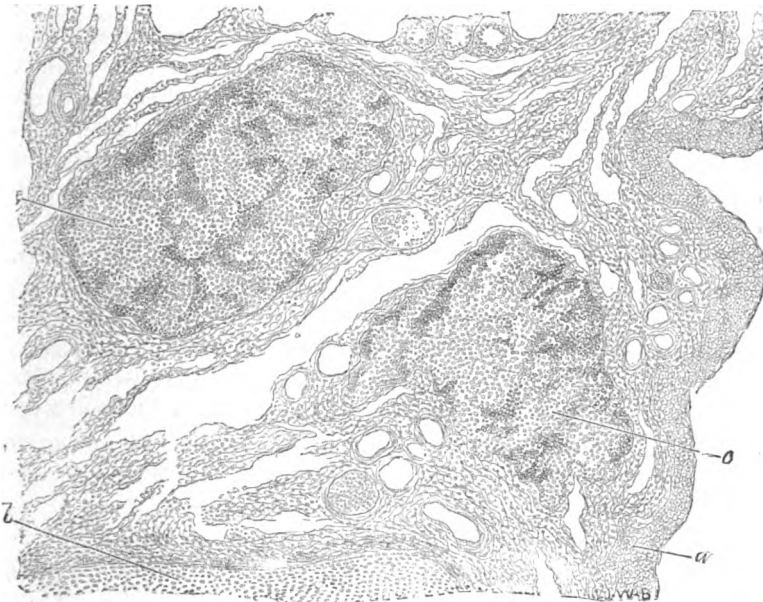
pathologists: while they are not directly involved in the tuberculization as a rule, they undergo, when implicated, two processes of infiltration simultaneously.

I. *Inter-acinous*; i. e., great increase of round-cells in the inter-acinous connective tissue; or infiltration between the acini.

II. *Intra-acinous*; i. e., interstitial increase of round-cells; infiltration within the acini.

The inter-acinous infiltrations separate the individual acini, and partially compress them. During this process, or somewhat later, miliary tubercles, likewise, collect both in the connective tissue between the individual glands, and between the individual acini of individual

Fig. 6.



Miliary tubercles in inter-arytenoid fold of an infant seven months old.¹ a, epithelium; b, right supra-arytenoid cartilage; c, tubercles.

glands. The gland-cells lining the proper membrane become detached and undergo destruction; and, as the membrane is forced inward by the external pressure exerted upon it, the diameters of the acini show their compression from globular into irregular oval, and elliptic bodies.

¹ This specimen shows, too, the layer of squamous epithelium at the posterior wall of the larynx.

Many acini finally undergo partial or complete destruction, it may be, from fatty degeneration following the combined internal and external pressure. The ducts (Heinze) resist the process longer than the acini.

Secondary tuberculosis of the larynx occurs, according to my own clinical observations, both in an acute and in a chronic form. The acute form occurs chiefly in cases of rapidly caseating pulmonary tuberculosis, is liable to occur quite early in the disease, and has an average life of from six to eighteen months. The several varieties of the more chronic forms occur chiefly in the more languid cases commencing as localized pneumonitis, occur at a comparatively advanced stage of the disease, and last from two to four years, or even longer.

The earliest recognizable stage of the *acute* form is almost always manifested by marked *congestion* of the mucous membrane. The earliest recognizable stage of the *chronic* and much more frequent form, is almost always manifested by marked *pallor* of the mucous membrane.

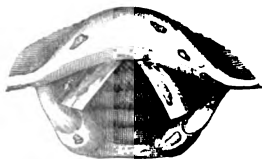
In the acute form.—The period of the pulmonary disease at which the secondary tuberculosis takes place is not uniform. In most instances, evidence of softening are indubitable when the laryngeal disease comes first under observation; but in many they are absent, or escape detection.

The intense catarrhal laryngitis of the acuter form usually subsides, in the course of from two to three weeks, into a severe chronic catarrhal laryngitis, indistinguishable, laryngoscopically, for a considerable period (perhaps as many as two to six weeks) as a malady due to specific constitutional disorder. In the course of from three to six weeks, a number of superficial ulcerations become noticeable upon the surface of the mucous membrane; most frequently upon the upper portion of the posterior surface of the epiglottis, frequently upon the inter-arytenoid fold, less frequently upon the inner face of the arytenoid or supra-arytenoid cartilages, upon the vocal bands, or upon the ventricular bands, and other portions of the interior of the larynx (Fig. 7). These

initial multiple superficial ulcerations of quasi-tuberculous origin may not be followed or succeeded by any other local manifestations characteristic of tuberculosis.

They present at first the closest physical similitude to the catarrhal epithelial erosions or aphthous ulcerations of a chronic laryngitis, with especially irritating secretory products. Suspicion as to their dependence upon

Fig. 7.



Multiple initial superficial ulcerations.

tuberculosis is excited by their multiple manifestation; purely catarrhal erosions being solitary, or very few in number.

The shape presented by these superficial ulcerations differs with the locality they occupy. They are roundish or ovoidal on the epiglottis, and upon the pharyngeal surface of the supra-arytenoids; elliptic or linear on the vocal bands and on the inter-arytenoid fold. This difference in configuration, being unusual in mere catarrhal erosions, is indicative of a difference in character. Catarrhal laryngitis coexists with the condition under consideration, as in several other varieties of intra-laryngeal disease: but the multiple ulcerations seem specifically due to the underlying dyscrasia, and to the attendant catarrh.

Erosions purely catarrhal in origin may also exist, but they are not essentially part and parcel of the tuberculous malady.

These individual ulcerations gradually extend in depth and in periphery, and often coalesce. Thus they can rarely be submitted, in their initial state, to the observation of the pathological anatomist.

Erosions similar to these, however, sometimes take place from time to time as fresh manifestations in the more chronic form of the affection; and these latter forms often come under post-mortem observation; as in the specimen herewith presented.

Inferentially it may be presumed that the early erosions in the acuter forms of laryngeal tuberculosis are similar to these in their histologic pathology.

The fresh superficial ulcerations last alluded to, exhibit under the microscope a loss of substance, confined to the epithelial layer in many examples, while extending in others to the immediately sub-epithelial portion of the mucosa also. Portions of epithelium implicated, but not yet exfoliated, are turbid; and, at some points, partially detached.

Ulcerations extending somewhat deeper into the mucosa show different stages of cell infiltration, erosion of vessels, accumulations of detritus, and fatty degeneration. *But no miliary tubercle can be detected either in the beds and edges of the ulcers, or in the tissues in their immediate vicinity.*

The broken-down *débris* of tubercle presenting nothing characteristic, the tuberculous nature of these initial superficial ulcerations is inferred, therefore, from the fact that they are rarely observed apart from subsequent undoubted tuberculous manifestations in different portions of the laryngeal mucous membrane. If this inference be justifiable, it should be admitted that tuberculous ulceration may occur independently of direct tuberculous infiltration *in situ*; in which case

they might probably be accounted for by so great a constitutional proclivity to tuberculous degeneration as to favor ulceration as a result of local irritation of almost any kind. Should this view of the subject be unsatisfactory, we would be obliged to regard these ulcerations as non-tuberculous in character ; or to acknowledge the probability of an actual infiltration with tubercle, and its rapid destruction and discharge. Later in the disease, when these ulcerations have extended in depth and in periphery, or by coalescence, their positive tuberculous character becomes manifest ; as is evident, after death, by the detection of secondary miliary tuberculosis in parts immediately contiguous.

As previously intimated, it is both a matter of doubt and of dispute, whether these shallow ulcerations are preceded by miliary tubercle. They come to the notice of the laryngoscopist, as a rule, only after the disease has made such progress as to have rendered an examination almost a matter of necessity ; and he misses the pre-existent lesion.

In this connection it will be useful to mention an instance in which Prof. Schnitzler, of the Vienna University, claims to have had, an opportunity, and the only one he has had, to watch the development of shallow ulcerations from miliary tubercles.¹ It is the most useful instance that I have come across in my reading ; although fully as authoritative assertions have been made by others as to having encountered cases in which they could detect the miliary tubercles laryngoscopically. These opportunities are so rare, however, that Tuereck never reported more than one in his famous clinical collection ; and Stoerck, of Vienna, now the oldest living laryngologist, has reported but one from his extensive experience.

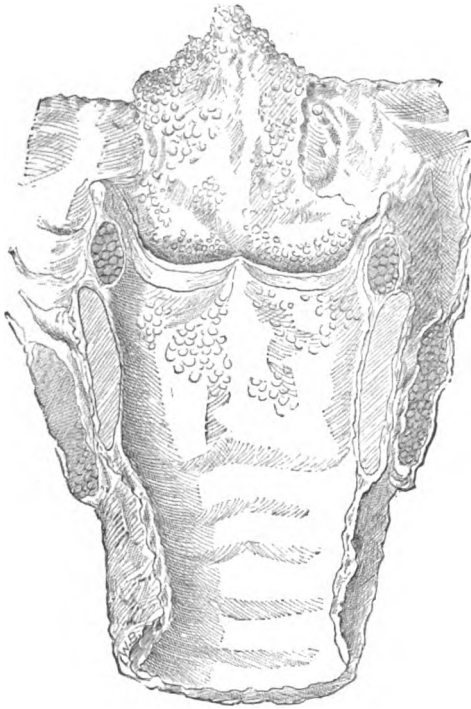
As for myself, I have never been able to detect tubercle laryngoscopically ; not even in the case of the preparation which I present, in which ample evidence of tuberculous infiltration was visible microscopically in the well-washed specimen (Fig. 8).

To resume the clinical pathology of laryngeal tuberculosis, it is to be remarked that subsequent laryngeal manifestations of local tuberculosis may be moderate or severe. As a rule, some characteristic circumscribed tumefactions attract notice not many days after the advent of these shallow ulcerations, and while they are increasing in depth and in periphery and by coalescence. Similar characteristic

¹ Zur Kenntniss der Miliartuberkulose des Kehlkopfes und des Rachens, Wien, 1881.

changes are manifested in some instances previously to any ulceration of the mucous membrane whatever. They take place at almost any stage of the malady; and they may remain the sole visible manifestation of the tuberculosis. These circumscribed tumefactions are chiefly thickenings of tissue, and are observed at various points of the structures.

Fig. 8.



Tuberculous infiltration of larynx.

The intumescence occurs chiefly in the mucous membrane of the epiglottis, ventricular bands and vocal bands; and less often in the inter-arytenoid fold and aryteno-epiglottic folds; in the last-named structures, perhaps, less frequently than in any others; presenting, in this respect, a marked contrast to the more chronic form of laryngeal tuberculosis, in which these selfsame aryteno-epiglottic folds are much more frequently involved than any of the other tissues.

The epiglottis undergoes tumefaction on its posterior aspect, to

several times its normal thickness; its functions as an obturator to the larynx in deglutition being thereby impeded.

The ventricular bands thicken so as to overlap the vocal bands to a considerable extent; and even to conceal them from view in some instances.

The vocal bands become thickened into veritable *vocal cords* indeed; and their inferior aspect becomes tumefied; sometimes to such an extent, that, as in a specimen exhibited by me before the Society,¹ some years ago, for the purpose of demonstrating the fact, they are transformed into thick, bulky, tumor-like folds, absolutely larger than the ventricular bands themselves.

When these tumefactions are multiple or bilateral, as often occurs, their encroachment upon the calibre of the larynx is in some instances so great as to produce a veritable stenosis, threatening asphyxia, and sometimes requiring tracheotomy.

Not only do these tumefactions occur with the ulcerations just discussed; but they also accompany other intra-laryngeal tuberculous processes. They may also take place without previous ulcerations or other visible manifestations of tuberculous disease.

The swollen mucous membrane becomes irregularly tumid, acquires a pallid yellowish-gray or gray color, as is observed in the early stage of the more chronic form, looks sodden and corrugated, and often supports a dingy yellowish pultaceous deposit.

Under the microscope, these swellings are found to be due to copious infiltration of small (lymphoid) cells from immediately beneath the epithelium inwards, both in the mucosa and in the sub-mucous connective tissue; massed here and there into tubercle nodules or groups of miliary tubercle, some of them undergoing central caseation. They extend from the epithelial layer as far as the glandular structure; the older tubercles being usually the deeper-seated ones. The glands, as a while ago stated, are not often themselves infiltrated, but the infiltration is usually massed around them and between their individual acini, which are thus compressed out of shape and subjected to fatty degeneration from pressure.

When the epithelium becomes detached, hemorrhagic ulcerations are thus exposed, of undoubted tuberculous character. These ulcers rapidly extend in the most irregular manner; so that large portions of tissue are soon included in their ravages. Their edges are well defined

¹ Trans. Path. Society, Phila., vol. v. p. 83.

and often slightly hemorrhagic or injected; their beds are rough and irregularly mamellonated, and usually covered with caseous detritus. The ulcers deepen and deepen in convergent outlines; or undermine the surrounding tissue at different points of their periphery. They extend to the ultimate limits of the cell infiltration, and may thus lay bare the very perichondrium—the first structure that seems fashioned to resist actual tuberculization.

Submitted to the microscope, these ulcerations usually present one of three conditions:—

I. No evidence of tubercle in either bed or edge of the ulcer, but infiltration of granular tubercle in immediate contiguity to the ulcer, or but a short distance from it.

II. Either infiltration of granular tubercle, or nodular tubercle in edges or bed, or both, without either contiguous or distant infiltration; and,

III. And most frequently, tubercle in the edges and bed of the ulcer, associated with granular tubercle-infiltration, whether contiguous only, or copiously disseminated throughout the mucous membrane.

As the disease progresses, nearly the whole interior of the larynx becomes involved. The tissues, generally, become so swollen that all the sharp outlines (edges of ary-epiglottic folds, vocal bands) become lost in thick welts. Later, as the ulceration extends, the whole structure becomes transformed into an irregular, excoriated, ulcerating, almost fungoid mass. Especially is this marked in the epiglottis and vocal bands; the latter, by fissure-like longitudinal ulcerations, becoming converted into a series of bands.

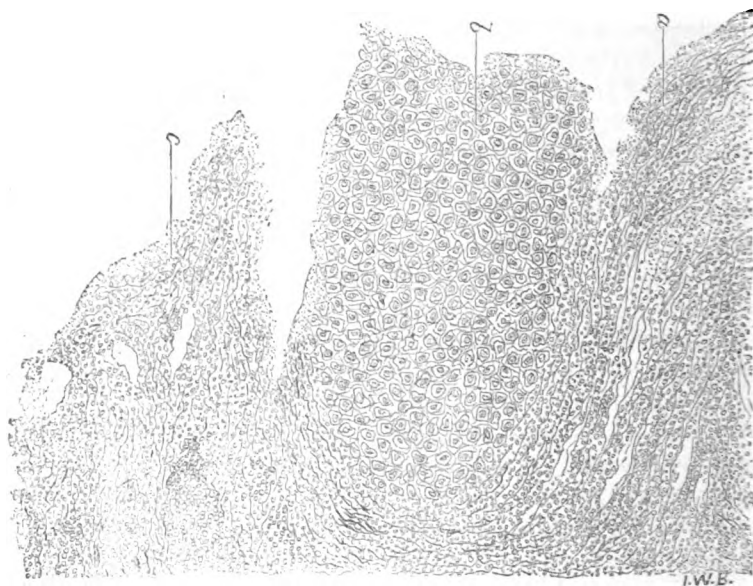
In many instances, local suppurative centres of inflammation become established in the immediate contiguity of these ulcerations, and, as they reach the perichondrium, involve that structure also, so that the cartilage becomes exposed.

The cartilage, too, becomes attacked, and undergoes destruction, in *débris*, as is most usual with the epiglottis (Fig. 9), or in fragments and in mass, as with the vocal processes, arytenoids and supra-arytenoids, and even the cricoid; or in large plates, as with the cricoid and thyroid cartilages. These fragments are often discharged by expectoration; the posterior vocal processes, and even the entire arytenoid cartilages, being, at times, exfoliated in mass. In the dead body, such fragments, or detached cartilages, are often seen loose in the abscess which surrounds them.

The epiglottis is sometimes destroyed in its entire free portion, by

progressive ulceration from above downward, so that a mere deformed stump remains; occasionally the destruction is by progressive ulceration from the side.

Fig. 9.



Tip of epiglottis, showing tuberculous ulceration and necrosis of cartilage, $\times 60$.
Section cut by Dr. Formad.

The similitude of the laryngeal pictures of this variety of laryngeal tuberculosis, to those presented in presumptive primary laryngeal tuberculosis, indicate to my mind such similitude in character that the latter may be regarded as acuter examples of the former. Both occur in cases of rapid pulmonary tuberculosis; both become associated with secondary tuberculosis of the trachea, pharynx, palate, tongue, lips, and other structures; both progress without interruptions.

The chronic form of tuberculosis of the larynx is not ushered in by congestion following distinct history of exposure, as in the acuter form just described; but it is characterized by well-marked pallor of the mucous membrane; a pallor participated in, in many instances, by the mucous membrane of the pharynx and mouth. This anemia is often apparent long in advance of similar evidence of impairment of nutrition elsewhere.

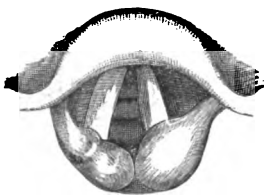
Although somewhat characteristic, and strongly suggestive of constitutional tuberculosis, this appearance is not peculiar to the tuberculous larynx only. In many cases, congestion ensues at a later period of the disease; as a result of the irritation produced by the tuberculous process;—but in many, the pallid hue continues to the very last.

Accompanying this local anæmia, there is an irregular vascularity of portions of the mucous membrane, which, in localities where its connections are loose (inter-arytenoid fold, ventricular bands), is elevated here and there in irregular wrinkled ridges or welts, red or gray in color, often of a distinctly villous aspect.

Some instances present spots of ecchymoses, or little irregular varices, irregularly located on the laryngeal surface of the epiglottis, or on points in the interior of the larynx proper. There is often a general tumid condition of the mucous membrane; but this is by far a less frequent condition than circumscribed tumefactions of a peculiar character.

I. The most frequent locality of tumefaction is in the tissue inclosing the supra-arytenoid cartilages and the apices of the arytenoid cartilages; the least frequent point of intumescence in the acute variety. These tissues, for the intumescence is most frequently bilateral, undergo gradual transformation into pale, tumid, club-shaped, and finally distinctly pyriform or irregular globose tumors, gradually tapering off,

Fig. 10.



Supra-arytenoid pyriform intumescence at an early stage.

Fig. 11.



Supra-arytenoid pyriform intumescence at a later stage; with thickening in the epiglottis likewise.

stem-like, toward the epiglottic extremity of the aryteno-epiglottic fold; with obliteration of all evidences of demarcation between the edge of the fold and the peculiar outline of the cartilages of Wrisberg and Santorini (Figs. 10, 11), and the intervening tissue.

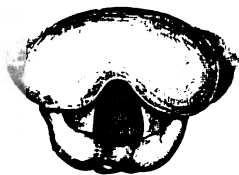
Although most frequently pale in color, these pyriform tumefactions sometimes become very much injected; sometimes intensely so; in

exceptional cases, to actual lividity at the points where they press against each other in the movements of phonation and deglutition.

The tumefaction, if unilateral, or the greater of the two, if bilateral, is, according to my own experience, almost always situated on that side of the body corresponding to the lung more advanced in disease. This peculiar pyriform aspect of the aryteno-epiglottic fold is sufficiently characteristic of coexistent pulmonary phthisis to establish the diagnosis. It occurs chiefly in slow cases, commencing with a localized pneumonitis; and is noticeable at a comparatively advanced period of the first stage of the disease.

It is a hyperplasia due to extensive proliferation of lymphoid cells in the adenoid tissue normally very abundant in this structure.¹ The increase of thickness of the mucous membrane is often three- or four-fold; so that it not infrequently measures fully one centimetre. Later in the disease, both granular and miliary tubercles are found. These swellings, as I have seen them, never subside, except in such partial measure as may have been due to effusion of serum or other products, as a result of the inflammatory process, set up mechanically by pressure, or by irritation. They are distinct from the slight serous oedema which sometimes accompanies protracted subacute laryngitis, with which they are occasionally confounded.

Fig. 12.



Turban-like thickening of the epiglottis.

Fig. 13.



Crescentically swollen epiglottis overhanging the orifice of the larynx.

II. In another group of cases the epiglottis undergoes great thickening of its free surface, from tuberculous infiltration, into a tumid ridge of turban-shape (Fig. 12); its edge often presenting as a thick crescentic cushion or pad, in some instances overhanging the laryngeal

¹ Wagner, Das tuberkelähnliche Lymphadenom. Leipzig, 1870-1.

orifice so as to conceal all the tissues from view, save, perhaps, the pyramidal intumescence of the supra-arytenoid cartilages (Fig. 13).

This may be associated with previously existing pyriform tumefaction of the supra-arytenoid and aryteno-epiglottic structures; or it may exist independently of any such manifestation at the time or at any subsequent period.

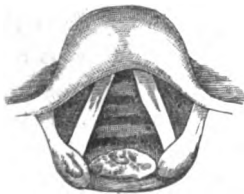
Ulceration usually begins superficially upon the laryngeal face near the edges, and gradually extends in depth and periphery. In most instances, progressive destruction takes place from above, until nothing but an irregular stump remains; but sometimes it proceeds from the side inwards, as admirably shown in the drawings exhibited.

As ulceration reaches the petiolus, it spreads along the commissure of the vocal bands into the ventricles, and along the ventricular bands.

Ulceration limited to the epiglottis is exceedingly infrequent, and is seen chiefly, if not only, in subjects who succumb rather early to the general malady. Ulceration of the anterior or lingual face of the epiglottis is unusual, and is almost always an extension of ulceration from the posterior face along the free edge. In one of the specimens under the microscope, however, the lingual face of the epiglottis is equally with the laryngeal face beset with tubercle infiltration.

III. Still another group of cases may be classified in which the epiglottis is flaccid instead of rigid; and in which the peculiar manifestations begin on the posterior portion of the larynx, and chiefly on the inner surfaces of the meso-arytenoid fold—a point of tissue which almost invariably suffers likewise in cases which have begun with other manifestations.

Fig. 14.



Condylomatous inter-arytenoid infiltration;
color normal; softening right apex.

Fig. 15.



Acuminated inter-arytenoid infiltration;
condensation right apex; red right vocal
band; thick and irregular vocal bands.

This fold of tissue becomes red, its normal surface interrupted, irregularly tumid in projections which often increase in size until they simulate polypoid excrescences, condylomatous (Fig. 14) or acumi-

nated (Fig. 15). Under the microscope, granular and nodular tubercles are found beneath the epithelium. Small superficial ulcerations soon appear in this fold, or upon these projections of the mucous membrane. These ulcerations increase in periphery, coalesce, and become covered with a yellowish, grayish detritus, which, when wiped off with a brush or sponge, or subjected to a douche or spray, reveals an irregular and slightly hemorrhagic surface; the ulcer having bled freely, though slightly, on contact with the cleansing substance. The surface is almost constantly covered with a thin layer of the products of secretion and disintegration.

In all these varieties of secondary tuberculosis of the larynx the normal color of the vocal bands is retained in most cases. In some they are injected, sometimes deeply, and in occasional instances are hemorrhagic on some points of their surface. But in almost every case the peculiar polish of the surface, which presents such a remarkable appearance in health is lost, and their aspect is dull.

In other cases, while the general surface of the vocal bands is congested, there are opalescent patches, dingy white in aspect, and more or less irregularly parallelogramic in configuration; probably groups of turbid pavement epithelia. Sometimes the smooth surface is interrupted by projections resembling excessive granulations. Ulceration takes place at the outer margins or edges of the vocal bands usually, and they become irregular in outline (Fig. 16); sometimes by small losses of substance, looking as though removed by a punch; sometimes in a jagged or more or less serrated edge.



Fig. 16.
Ulcerated vocal bands and ventricular bands, cavity left apex; solidification of right apex.

The breadth of the band is thus actually diminished at the points where ulceration exists, and often looks still more attenuated in consequence of the overhanging of the ventricular band. These ulcerations are most frequently seen at or towards the posterior extremities of the structures. Should the vocal band give way in great extent, as occasionally happens, retraction occurs in the fragments; and the shape of the glottis becomes, in consequence, very irregular.

Should it give way at the posterior vocal process, that structure will be apt to project across the glottis.

Fungous vegetations are sometimes developed upon the ulcerated

edges of the vocal bands; and these sometimes produce adhesions at the anterior portion of the bands.

Collections of tubercle within diffuse tuberculous infiltration is rarely observed in the mucous membrane of the vocal bands. It is infrequent also in the elastic fibrous tissue in the muscular substance, or between the muscular fibres (Heinze).

IV. Still a series of cases may be differentiated in which the principal lesions are located in the vocal bands themselves.

The posterior surfaces of the vocal bands undergo great intumescence, so that they project beyond the vibrating edge of the band, and encroach seriously upon the calibre of the larynx. The dyspnoea from this stenotic condition is often sufficiently intense to threaten asphyxia, and only to be alleviated by tracheotomy.

Ulceration usually takes place in the longitudinal direction of the bands, and the irregular appearance is quite suggestive of laryngeal neoplasms.

Tubercle is infrequent either in the muscular substance of the vocal band or between the muscular fibres.

Pyriform intumescence of the posterior portions of the aryteno-epiglottic folds has, in my practice, been far more frequent in young adults than in those in middle life; but I have seen the condition in patients as old as fifty-six.

Patients above fifty years of age are much more apt to exhibit, in my experience, torpid ulceration, with imperfect granulations, on the inner aspect of the mucous membrane over the supra-arytenoid and arytenoid cartilages; and most of the cases I have seen have exhibited both the ulceration and the pulmonary softening on the left side.

Perichondritis and chondritis, which usually attend the later stages of the malady in prolonged cases, are to be regarded as inflammatory processes of septic origin due to the tuberculosis; and not as tuberculous processes proper. Tubercle is rarely found close to the perichondrium; the infiltration, as has been stated, being chiefly in the upper part of the mucosa.

These processes may be limited in extent, be confined to one surface; or they may be extensive and involve the entire surface, leading to proportionate destruction of tissue. But the processes themselves are by no means always proportionate to the intensity of the tuberculous process which has excited them.

Abscesses often result from the perichondritis, and usually point inwards towards the free surface of the larynx. They are liable to be

productive of suffocative phenomena. Their incision and discharge, when practicable, usually relieves the threatening asphyxia, which, under other circumstances, may compel resort to tracheotomy.

After intra-laryngeal discharge of the abscess, spontaneous or artificial, fragments of cartilage are sometimes seen projecting into the free lumen of the larynx. This takes place most frequently with the posterior vocal processes, the arytenoid and supra-arytenoid cartilages, and much less frequently with the cricoid cartilage.

Tuberculous ulcers rarely heal. Exceptional cases of cure are occasionally noticed, but it becomes questionable whether those instances have been examples of actual tuberculous ulceration; and the conservation of the patient's life prevents an accurate answer to the query, for verification is possible only under microscopic inspection.

A disposition to heal is sometimes manifested by the appearance of healthy looking granulations upon the bed of the ulcer; but ere long the tuberculous infiltration occurs in the new-formed tissue which speedily succumbs to the invasion. Exuberant granulations are often developed in these ulcers, and even proliferate at times into veritable tumors or vegetations, which sometimes require removal to clear the air-passage from obstruction to respiration.

In cases of pulmonary tuberculosis following as a sequel to caseous pneumonitis, the general vitality of the tissues is impaired to such a degree that even follicular ulcerations of the larynx are insusceptible of cure.

The percentages of cases of pulmonary tuberculosis in which the larynx undergoes tuberculization has not been approximatively estimated; but it is probably not very large, and is much greater in hospital than in private practice. Heinze's personal pathological investigations yielded a percentage of 6.5. Men are more liable than women, in the proportion of nearly three to one; probably as the result of their greater exposure to inclement weather.

As mentioned earlier, the disease appears to be more frequently encountered in inherited than in acquired pulmonary tuberculosis.

The period at which tuberculization of the larynx occurs is variable, but as a rule the disease in the lungs is more advanced than in the larynx. If tuberculous ulceration has taken place in the larynx, cavities are almost always to be found in the lungs.

Inherited or acquired tendency to catarrhal inflammations of mucous membranes, especially to inflammations of the mucous membranes of the aerial tract, seems to be a causal factor in the development of

tuberculosis of the larynx. Inheritance, in my experience, has seemed to be an important factor.

The tuberculous process itself seems to be an inflammatory process attended with profuse cell-proliferation, in tissues abundantly supplied with blood and lymph channels; the proliferation being too profuse for absorption, and undergoing maceration or decay.

Some acute observations of Dr. Formad appear to indicate an anatomical reason for the development of tubercle in certain subjects, and I trust that he will present these views to us this evening. Suffice it now to say, that he finds the lymph spaces extremely small in those animals most readily tuberculized, and likewise in the tuberculized subjects of the human species, and in tuberculous subjects only.

During the last three years I have, as opportunity permitted, examined the blood of many of my cases of tuberculosis of the lungs and larynx. While often failing to encounter any appearance at all peculiar, I have so frequently observed a condition which has not been noticed in my examinations of blood in connection with other disease, that it seems to me to have some very close relation with tuberculosis. In all the cases with a temperature exceeding 100° F., this condition was almost sure to be found, and was rarely encountered at a lower body temperature.

In the first place, the proportionate number of white corpuscles is much diminished; secondly, the red corpuscles have a great tendency to become massed; and, thirdly and chiefly, plugs of granular matter become caught here and there between the individual members of certain groups of red corpuscles.

These granules look like granules which may have escaped from the white corpuscles, perhaps by dissolution of the protoplasm; and this of course would account for the paucity of the white corpuscles.

Clinicians have been too much inclined to regard the morbid manifestations which have been under consideration, as pathological processes remotely due to tuberculosis, but not in themselves tuberculous nor confined to tuberculosis.

Pathologists, with but few notable exceptions, view them as specific results of tubercle, the direct outcome of tuberculization, and not the mere result of irritation occurring in tuberculosis. The tenor of these remarks would indicate the greater confidence in the latter opinion.¹

¹ Every point touched upon in the foregoing paper was amply illustrated by specimens, microscopic sections, and drawings. The various sections under the microscope were prepared by Drs. Longstreth, Seiler, and Formad, from

Dr. Tyson said that if there had been any doubt in the past as to the occurrence of laryngeal tubercle, the very graphic illustrations shown by the lecturer, as well as the microscopic sections exhibited by him, placed the matter beyond question. It now only remains for future pathologists and clinicians to determine the relation between laryngeal and pulmonary tuberculosis. Owing to the fact that only by microscopic sections can the presence of tubercle be absolutely determined, and that when an autopsy is obtainable, secondary changes have complicated the primary affection, the question seems almost impossible to decide, and its answer becomes merely a question of probability. He would ask the lecturer whether it was possible by the laryngoscope to diagnosticate tubercular ulceration from follicular ulceration of the larynx? Again, what proportion of the laryngeal ulcerations occurring in phthisis are tubercular, and how often are such cases merely follicular ulcerations? Finally, as a matter of great importance both for prognosis and treatment, can the syphilitic ulcer of the larynx be readily distinguished from the tubercular?

Dr. Seiler said that he had for a long time been specially interested in the subject of laryngeal tubercle, and when working more than at present with the microscope, had devoted much time to the practical investigation of the matter. Although he agreed with Dr. Cohen as to the impossibility of absolutely determining by the laryngoscope the presence of tubercle, yet he had no doubt that it was present in a large proportion of the cases, both of acute and chronic phthisis. Microscopically, tubercle of the larynx rarely shows the presence of giant-cells, but Dr. Seiler had most commonly noted depots of small round cells between the glands, whose centres became caseated, and also that the same infiltrate affected the glands, likewise undergoing later on caseation. He must take exception to one statement of the lecturer, viz., that the pyriform swelling of the arytenoid cartilages "*never* disappear," since he had had at least five cases under careful observation for years, where such disappearance *had* occurred. He had nothing further to add with regard to this admirable paper, which embodied more fully and correctly all the facts relating to laryngeal tubercle than any other which he had either heard or read.

Dr. Eskridge remarked that Austin Flint, in his clinical work on Phthisis, states that laryngitis was found in a very small proportion of

three of the specimens exhibited; and they represented tubercle infiltration in the various component tissues of the larynx. The camera-lucida drawings were made by Dr. Blackburn, under the immediate supervision of Dr. Formad.

the nearly six hundred cases of consumption observed by him. According to the author, no laryngoscopic examination was made, the presence of hoarseness being relied upon for the recognition of laryngeal involvement. He would ask the lecturer whether he had seen any cases of tubercular laryngitis in which hoarseness did not exist. Dr. Eskridge then referred to Koch's theory, and said that if tuberculosis were due to the inhalation of bacilli, the nasal cavities ought to be affected. He would like to know whether the author of the very excellent paper for the evening had met with a case of tubercle of the nasal passages, especially of the posterior nares.

Dr. J. C. Wilson asked whether cicatrices or adhesions occurring in connection with ulceration at other points, might not prove diagnostic between syphilitic and tubercular ulcerations.

Dr. Cohen, in closing the debate, said that he must first express his thanks to Dr. Formad for his kindness in supervising the preparation of the numerous microscopical specimens and drawings thereof with which he had illustrated his paper. In reply to Dr. Seiler's cases of cures of the pyramidal swelling preceding or accompanying laryngeal tubercle, he could only say that they were decidedly exceptional. It would be recalled that he had distinctly stated in his paper that when the swelling was partly due to œdema and effusion of lymph it might of course disappear. To Dr. Eskridge's question, he would reply that hoarseness does not occur in the majority of cases, except where the vocal cords become distinctly involved on the one hand, or their movements become impeded by swelling in other tissues on the other. He knew nothing practically concerning bacteria, but as he understood the theory, it was only where they were arrested and detained in the laryngeal secretions that infection took place. If he had seen cases of tubercle of the nasal passages, he had certainly never recognized them as such. As to the possibility of distinguishing between tubercle and follicular ulceration of the larynx by the laryngoscope, he must confess his inability to point out any diagnostic points, although he thought that it could be done, predicated on the general appearance of the parts and the general condition of the patient. As with these two affections so with syphilis. There are peculiarities in edge and outline, but he would be unwilling to say from the appearances alone whether a given case was or was not specific. It has been said that the syphilitic ulcer progresses from above downward, while the tubercular pursues a reverse course; but some of the cases detailed this evening prove that tubercle sometimes progresses from above likewise.

Dr. Cohen thought that the specific ulcerations were more apt to be

symmetrical. The patient's history, and the influence of specific remedies often determined the diagnosis. In reply to Dr. Wilson, he would say that in syphilis cicatrices were far more apt to be present, while, on the contrary, you rarely see even an attempt at healing in tuberculosis of the larynx. The therapeutic test, however, was the most reliable.

September 28th, 1882.

6. *Elephantiasis græcorum of the tongue and larynx.*

Presented by Dr. A. C. W. BEECHER.

The case from which the accompanying specimen was taken, was reported in the *Photographic Review of Medicine and Surgery*, No. 6, vol. i., August, 1871 (Drs. Maury and Duhring), Philadelphia.

Mr. —, æt. 26, born in Cuba of Spanish parentage, married. His father was still living when the subject of these notes died in 1872; mother died when he was an infant. He was nursed from the breast of a house-servant, a negress, who was unmarried, but had had several children by different individuals. As far as known, she was healthy, with the exception of sores upon her feet. He had none of the diseases of early childhood excepting mumps and measles. He enjoyed good health until he was fifteen years of age, when spots appeared upon his body; these were yellowish-pink in color, and very abundant, seeming to be just beneath the skin. There was neither pain nor itching in them. They remained about one year, and disappeared during a voyage to Spain. While in Spain he had an attack of neuralgia in the little finger of the left hand, running along the course of the ulnar nerve to the elbow. This was relieved, and he returned to the West Indies. Six months after this he had another attack of the same trouble, which, however, lasted one month. In 1862 he came to this country to complete his education. Having been here about one year, he became subject to catarrhs, which would centre about his throat and cause him annoyance. Soon after this he was conscious of itching in both arms, from the elbows to the hands, and in his legs from the thighs to the feet. In 1866 his hands and feet began to swell as if œdematous, and this would extend no further than the wrists and ankles, producing a sensation of stiffness and inability to move the fingers and toes. While the hands were so swollen, blebs of various sizes appeared suddenly upon the dorsum of the hands and

fingers, extending over their whole length. At times he would be aroused at night with a sharp pain in the hands and fingers, and in the morning would be surprised to find a large blister, which had come on through the night. These bullæ contained a whitish opaque fluid, and upon bursting would be followed by scabs, which were dark-brown in color and transversely cracked and fissured over the joints; underneath these crusts were excavated ulcers. The tips of the fingers became involved, having similar ulcers, the nails growing over them, and breaking off when long. The feet were similarly affected. Following this eruption, he had frequent hemorrhages from the nose, coming on while in hearty laughter or in mental or physical excitement. The nose now became tender, was much swollen, and discharged matter, which was not very offensive; at the same time the nose began sinking to its present condition. The diseased throat and nares were at no time entirely relieved. About a year after the first outbreak upon his hands, his face was attacked in the same manner with vesicles and ulcers. These healed slowly, and left the cicatrices which are now visible. As rapidly as some healed others formed; and this process has gone on until the present time.

Present condition.—The body appears much wasted, and the skin is of a dusky hue. The hair of the body is scanty, and in many places absent altogether. The muscles of the arms are much atrophied, and small white cicatrices are visible on both arms and forearms. Upon both arms over the olecranon process, are situated large nodules, hard and firm, reddish in color. Similar nodules exist over the patellæ. The hands are much deformed. The little finger of the right hand is contracted upon the palm, and at the same time the first phalanx is extended; the nail has fallen off, and there is ulceration of the end. The other fingers are similarly affected. The dorsum of the hand is wrinkled, hard, and dry, with small tubercles scattered beneath the surface of the skin. A large ulcer exists over the styloid process of the ulna. Long, irregular, black scabs cover the dorsum of the fingers, under which exist ulcers. The left hand is more deformed than the right. Here the little finger is atrophied, and shrunken away to less than half its normal size. Ulcers exist upon all the fingers of the hand. A point of interest is that the metacarpo-phalangeal articulation of the thumb projects inwards towards the palm, the thumb being drawn over the palm. There is great impairment of function in all the fingers. Tortuosity and varicosity of the superficial veins about the wrists exist to a marked degree. The hair of the

head is normal in quantity, but is harsh, hard, and straight. There is total loss of the eyebrows and eyelashes. The hair about the face is thin and scanty. There is a large, hard tubercle over the frontal boss, and beside it an ulcer which has existed for some time. Below the nasal bones, the nose has sunken, and cicatrices exist over the entire face. Upon the right side of the face, over the zygoma, is an ulcer covered with a large black crust resembling rupia. The ears are very irregular in shape, and bear the marks of ulceration. Many of the teeth are decayed; and broken off near the gums, while the tongue is thickened, deeply furred, and the papillæ greatly enlarged. The velum palati is almost gone from ulcerative action, and yellowish-white tubercles exist in the pillars of the fauces. The larynx is much contracted by thickening of the mucous membrane. The epiglottis is about twice its normal thickness, and has several well-marked tubercles upon it, and also about its margin. The voice is much impaired; it is very limited in volume, and possesses a peculiar huskiness, though entirely free from nasal sound. Over the tendo Achillis of both limbs are large, deep ulcers, which have a sloughing, gangrenous appearance. The feet resemble the hands as regards ulceration and deformity, though to a less severe extent.

Sensation is very much impaired at certain portions of the body. When touched upon the back of the hand, there is only a slight appreciation, and it is referred, not to the point of contact, but somewhere in the neighborhood. The sense of pain is almost entirely absent in the hands and forearms, but becomes gradually appreciable as the trunk is approached. The patient can tell the difference of temperature between very hot water and water of ordinary temperature, and, moreover, his whole surface is rather sensitive to changes of temperature.

A pin passed through the pulp of the middle finger gave not the slightest pain. Turning the patient's head away, a lighted taper was applied to one of the fingers, and though a blister and charring of the part rapidly followed, yet there was no appreciation by the sense of pain. Sensation about the trunk is almost normal. Taste and smell are somewhat impaired. Sight is quite good, though he cannot use the eyes for any length of time without pain. The general health is much better than would be supposed, considering the case. There appears to be no trace of venereal disease. Sexual powers absent. He has frequent neuralgic pains shooting along the nerves in the arms and legs. General atrophy of the fingers, hands, and feet is now

going on, and it is appreciable from month to month, though his general health has changed but little since he came into my hands (Feb. 1871).

The foregoing symptoms continued. The corneæ became ulcerated, the conjunctivæ of the eyelids became agglutinated to the eyeballs, and almost total blindness ensued. The thickening and ulceration of the epiglottis made it almost impossible for him to swallow on account of the pain from the ulcers, and by the non-closure of the aperture of the glottis portions of the food would enter the larynx, producing violent and exhaustive coughing. The narrowing of the vocal box interfered with his respiration, as it had done with his speech long before. Thus he became extremely emaciated, and died Oct. 29, 1872, from partial starvation and partial suffocation, suffering to the last with greatest intensity. His sexual power and desire had been absent a long time before his death.

Autopsy thirty hours after death.—Rigor mortis well marked. Great emaciation of the entire body. Surface of tongue fissured, papillæ enlarged, remnants of tubercles well marked toward its base. The epiglottis is curved on its long diameter, is thickened to from one-sixth to one-fifth inch, and stiff; the upper margin is eroded by a large ulcer. A deep ulcer exists in the mucous membrane on the left side near the apex of the greater cornua of the hyoid bone. The thickening of the tissues and mucous membrane extends into the upper part of the trachea producing constriction of the aperture of the glottis and of the space between the vocal cords so that an ordinary quill could not be inserted between them. Sections were made of the ulnar nerves of both arms near the elbows and extensive degeneration was found. No other organs were examined.

This case was seen and diagnosed, by Dr. L. A. Duhring and confirmed by Dr. R. M. Bertholet, who made a laryngoscopic examination, to be of the mixed variety, viz., tubercular and anæsthetic leprosy. It is interesting from two standpoints beyond the fact of its being a rare disease among us, and the first of these is, the possibility of other cases being observed here since the disease has appeared in California, brought there by the Chinese, it being also present in the Province of New Brunswick, and in the West Indies. The conveniences of communication with these places may be the means of bringing other cases to this city.

The second point of interest is its resemblance to syphilis, into which error I fell in this case, while recognizing that there was

something strange about it. I asked the late Dr. F. F. Maury to see the case with me; he, too, pronounced it syphilitic. Subsequently Dr. Duhring saw the case and conclusively showed it not to be syphilitic but leprous.

Erasmus Wilson says: "The resemblance to secondary syphilis is so striking that an error is certain, excepting on the part of those who have had an opportunity of seeing and observing leprosy."

Hillis says:¹ "The mucous membrane inside the cheeks, nose, fauces, and tongue may be seen studded with tubercles; the tongue is swollen, fissured, denuded in parts of epithelium, the papillæ very prominent, and the organ sometimes presents two or more large globular tuberculated swellings, red, tense, and shining, around which smaller ones may be seen, some in process of ulceration. The velum palati may be lost, as in syphilis, but this occurs only when the disease has existed for three or four years" (p. 20).

Hillis relates a case and gives an illustration of a Chinaman with "mixed tuberculated lepra simulating chronic syphilis." "He is covered all over with tubercular infiltration and minute tubercles, which would require to be carefully diagnosed from syphilis, *but there is at present anæsthesia, which is not the case in the latter affection*" (p. 134).

Erasmus Wilson states that leprosy "resembles the zymotic affections and syphilis," and also "resembles these diseases in some of its phenomena."

Erasmus Wilson² records a case of "Elephantiasis anæsthetica, occurring with syphilis;" this case, after having been treated for secondary syphilis in India, came to England and was treated for the same trouble. Dr. Wilson, upon seeing the case, recognized its leprous character, which was confirmed by Dr. Boeck, of Christiana, Norway, who was at the time in London (p. 240).

Dr. Wilson records another case of "Elephantiasis anæsthetica; mistaken for secondary syphilis" (p. 242).

Dr. Bowerbank, of Kingston, Jamaica, says: "I believe it to be a disease sui generis;" "I have little doubt that leprosy and syphilis may run their course together" (p. 12).

"The peculiar snuffling so common among lepers from implication

¹ Leprosy in British Guiana. By John D. Hillis, F.R.C.S., M.R.I.A., London, 1881.

² Report on Leprosy by the Royal College of Physicians, London, 1867.

of the Schneiderian membrane, and the croaking voice from the vocal cords being affected, now become markedly characteristic, more so as the disease progresses. Speaking through the nose may be complicated with difficulty of breathing, from tubercular obstruction, and bleeding from the nose occasionally occurs."

"After death, I have found the true vocal cords and the epiglottis ulcerated, and the seat of tubercles." (Hillis, p. 20.)

"Besides the skin, the mucous membrane of the nose, fauces, larynx, hard palate, and tongue, the trachea, and large bronchi, have been found after death studded with various sized tubercles. They sufficiently account for the difficulty of breathing, hoarseness, or croaking, so common in advanced tubercular leprosy; and when they ulcerate, as they frequently do, the peculiar breath-fetor, so characteristic of lepers, results, as well as the other serious constitutional disturbances." (Hillis, p. 67.)

"One of my patients, a Chinese," had "obstruction to his breathing from tuberculation. He died, and on examination the glottis was found almost closed with tubercular thickening." (Ibid.)

"Amyloid degeneration will frequently be found in the liver, spleen, and kidneys; no doubt in consequence of the exhausting discharges from the extensive ulcerations." (Ibid.)

Hillis states that the parasitic theory of leprosy was first advocated by Dr. G. A. Hansen, of Bergen, in *Nodiskt Mediciniskt Arkiv*, Band i., No. 13, 1869, and Band ii., No. 16, 1870; also, in vol. i., *Med.-Chir. Rev.* for 1875. His discoveries related to the presence of bacteria or micrococci wherever the leprosy deposits were found.

At page 489 in the article just alluded to, Hansen thus makes reference to these: "There are to be found in every leprosy tubercle extirpated from a living individual small staff-like bodies, much resembling bacteria, lying between their cells; not in all, but in many of them. Though unable to discover any difference between these bodies and true bacteria, I will not venture to declare them identical. Further, while it seems evident that these low forms of organic life engender some of the most acute infectious diseases, the attributing of the origin of such a chronic disease as leprosy apparently to the same matter must of course be attended with still greater doubt. It is worthy of notice, however, that the large brown elements found in all leprosy proliferations in advanced stages . . . bear a striking likeness to bacteria in certain stages of development, as these are represented by Klebs." (Hillis, p. 142.)

Dr. Carter also refers to these bodies in the Transactions of the Pathological Society of London. (Vol. xxvii. p. 301.)

*The bacillus of leprosy.*¹—The evidence that we may safely speak of a bacillus lepræ, becomes daily stronger. Neisser, Eklund, Gaucher, and Hillairet confirmed the observations of Hansen; whilst, quite recently, Cornil and Suchard in France, and Majocchi and Pellizzari in Italy have given excellent descriptions and drawings of the parasite, which accord in every particular with those of Hansen. Prof. Köbner, of Berlin, has still more recently published an account of attempts made to inoculate animals with the disease, and in his suggestive paper the existence of the bacillus in leprosy tissues receives still further confirmation. It would seem as if the time would soon come when the development of leprosy will be found to be associated with the development of the bacillus lepræ in the vascular organs.

There is the question how the parasite obtains admission into the system, and, intimately associated with it, the debated subject of the contagion of leprosy.

The existence of bacilli in the circulating fluid affords a satisfactory explanation of the outbreak of leprosy in different parts of the body of the same person. How the bacillus obtains admission to the circulating fluid of an individual is a question that presses for an answer.

Bacilli are not found in the epidermis.²

Dr. Wilson asked whether the family history had been investigated.

Dr. Beecher replied that he had carefully questioned all concerned, but had ascertained nothing special, except that when such cases occurred in wealthy families, they were usually ignored, and the whole matter "hushed up."

Dr. Wilson said that in this connection he would call the attention of the members of this Society to a very able paper in the last issue of Hay's Journal, where the writer took the view that this disease was contagious, maintaining that to elucidate this point it should be investigated where it was of rare and recent occurrence. Dr. Wilson also referred to its occurrence among recent immigrants in certain of our Northwestern States.

Dr. Carl Seiler said that Dr. Beecher had asked him to examine

¹ British Medical Journal, Jan. 29, 1882, p. 174.

² Ibid., p. 175.

the specimen of the larynx and tongue of the case of elephantiasis, and that he had found the following lesions: The tongue was deeply fissured on its dorsum and the papillæ appeared enlarged. The epiglottis was thickened, very stiff and rolled on its long axis like a dry leaf. On its upper free margin was a crescent-shaped ulcer with raised edges, and numerous smaller roundish ulcers were scattered over the laryngeal surface of the epiglottis. Extensive ulceration of both the ventricular bands and vocal cords on both sides existed, so that the opening of the ventricle was almost entirely occluded. These ulcerations were symmetrical on both sides and most marked on the anterior insertion of the vocal cords. About a quarter of an inch below the cords was a cicatricial band projecting from the sides of the subglottic cavity and leaving an elliptical opening through which a crow-quill could barely be passed. Below this obstruction the mucous membrane of the subglottic cavity was studded with small, round ulcers, while the trachea seemed normal. Further lesions were not apparent to the naked eye, but he thought that others might have existed which had become obscured by the long maceration in alcohol to which the specimen had been subjected.

The Doctor said further, that it was a pity that the record of a laryngoscopic examination, which he understood had been made before the patient's death, had been lost, still the lesions seen in the specimen would explain the symptoms of dysphagia, aphonia, and dyspnoea, and it was astonishing how the patient could have carried on respiration at all through the narrow opening left by the cicatricial tissue below the glottis. The chief interest, however, centred in the great similarity of the lesions in this unique specimen with those found in syphilis and lupus of the larynx. He had seen ulcerations in syphilitic laryngitis almost identical in shape and location with those seen in the specimen, and remembered having seen two or three specimens of lupous ulcerations of the larynx when he was in Vienna which bore a strong resemblance to them. It was his opinion that by a mere laryngoscopic examination syphilis, lupus, and leprosy of the larynx could not be diagnosed from each other, but that other signs and symptoms outside of the larynx had to aid in the diagnosis. Thus in syphilitic laryngitis there were always sharply defined and abruptly ending bands of a deep red color on the free margin of the velum palati. In lupus, affections of the skin on some part of the body always preceded, co-existed with, or shortly followed the manifestation of the disease in the

larynx ; while in leprosy the larynx was usually attacked later in the disease, when other portions of the body clearly showed the marks of the pest.

Dr. Little remarked that, having conversed with Dr. F. H. Enders, who had seen a great many cases of leprosy in the Sandwich Islands, he had been interested to note that the eyelids were affected at an early stage ; ectropion resulting, and the conjunctiva and cornea or even the whole eyeball becoming involved. The affection of the eyelid is sometimes the first symptom to occur during the first or second year of the malady. The lids were involved in the case described, and the eyeballs subsequently.

November 23d, 1882.

7. *Secondary sarcoma of the heart, lungs, and gall-bladder, following amputation for primary deposit in the femur.*

Presented by Dr. DE FOREST WILLARD.

The specimens exhibited were the heart, lungs, and gall-bladder of a female patient 21 years of age, whose right thigh had been amputated four months previously for a spindle-celled sarcoma of the lower end of the femur.

The walls at the apex of the right ventricle were infiltrated with a sarcomatous mass, which extended into the cavity among the columns, forming an irregular-shaped body occupying one-fifth of the space. The walls were softened and but little of the muscular fibre was to be seen at the apical region. The diseased tissue was very soft and easily detachable, rendering its propulsion into the lungs a matter of exceeding probability at each heart beat. The walls above the mass were normal in appearance and in thickness ; the valves showed no evidence of disease on either side of the heart. The left ventricular and both auricular walls were healthy. The disease had not reached the visceral layer of the pericardium, and there was no abnormal effusion in the cavity of the sac. The septum ventriculorum was not involved.

That numerous particles had been swept into the lungs was very evident when these organs were examined. At a large number of points in either lung were to be seen white masses varying in size from that of a pin's head to that of an English walnut. Some of these were dense, others were undergoing softening, and in nearly every

instance the lung substance surrounding was so disintegrated that the mass seemed to lie in a cavity containing a drachm or more of sanguinolent fluid. A very moderate degree of pressure would cause a nodule near the surface to burst its pleural covering and give rise to an accident similar to the one which was found to have occurred near the right apex. At this point a large sarcomatous mass had excited a degree of inflammation sufficient to fasten the lung to the parietal pleura, and, one week before the patient's death, ulcerating through the serous covering, had given rise to an internal hemorrhage which was well nigh fatal, and which had produced the symptoms of sudden collapse noted in the history.

This escaped blood was found in the right pleural cavity, being confined by adhesions chiefly to the upper portion of the chest, and in the week which elapsed between the hemorrhage and death, had coagulated and contracted so as to form decolorized chicken-fat clots and other coagula to the amount of at least two pounds in weight. The pleural cavity below the adhesions contained about two quarts of bloody serum. There was no consolidation of the lung save around the diseased foci.

The lungs had evidently acted as a complete sieve and had prevented the passage of the poison, for liver, kidneys, spleen, and all the other organs were free from nodules save one small spot in the gall-bladder. The brain was not examined. Further infiltration of the body might have occurred at a later stage had not the above-mentioned accident caused the early death of the patient.

The primary disease in the femur had been developed into activity apparently as the result of traumatism, since no difficulty had existed previous to the reception of a severe injury of the knee, occasioned by falling upon the limb. From this time the pain on walking was continuous, and four months later there was decided enlargement of the external condyle and in the popliteal space.

The clinical history presented many interesting points during the eleven months in which the disease ran its course, but the most interesting ones were—

1. The lighting up of the disease by the injury, there being no known trouble previously, although nothing could be learned of her antecedent history.
2. The fact that the physician who first saw her could discover neither fracture nor luxation nor any displacement of cartilages, conditions which subsequent examinations would have tended to verify.
3. The appearance, about four months later, of a pulsating tumor in

the popliteal space, which tumor also possessed a decided bruit but no thrill. This was found at the post-amputation examination to be due simply to the lifting of the artery from its bed by the sarcomatous nodules, the artery not being even dilated.

4. The non-involvement of the knee-joint in any degree, although the nodules pushing through posteriorly between the condyles had even raised the synovial membrane. The cartilages covering the femur also were mere shells, the ravages of the disease having destroyed all the underlying bone tissue. At about the line of epiphyseal junction the entire extent of the femur was destroyed.

5. The return of the disease, not in the stump, but first in the right ventricle of the heart, giving rise to no dyspnœa but to failure in circulation and great prostration, which came on from four to six weeks after the amputation and without anything in the condition of the stump to warrant such depression; in fact, there was at no time in the rapid and satisfactory healing of the flaps any inflammatory action. The patient at this time seemed in articulo mortis, yet there was no pain and no dyspnœa, only a feeble, rapid heart action accompanied by low delirium and weakness. There were no valve sounds audible. Looking at the post-mortem revelations in the heart, it would seem probable that the depression was due to the taking root and formation of this secondary sarcomatous mass in the heart, overpowering and well-nigh causing cessation of its activity, yet so great was the accommodating power of nature that finally becoming accustomed to the at first unusual growth, the organ began slowly to adjust itself to the new order of things, and the patient began to rally. So great was the improvement, that three and a half months after the operation she was able to walk about on crutches, to ride out, eat heartily, and to consider herself almost well again. Adipose tissue had accumulated rapidly, there was only slight dyspnœa on exertion, she coughed no more than three or four times during the twenty-four hours, and then only slightly; there were no signs either in the symptoms or in the facial expression to indicate other than a slow return of the expected disease.

6. Suddenly, and without any causative exertion, the hemorrhage alluded to occurred, and although she recovered from the immediate collapse, yet she lived for one week, suffering no pain, and with only a moderate degree of difficulty in breathing, the respirations varying from 30 to 36; the pulse varying from 130 to 140.

7. The primary disease in the femur showed a preponderance of

spindle-cells not only in the soft cerebral-like tissue found in the bone itself, but also in the hard nodules surrounding the condyles; in the secondary growths the round cells were in excess of the spindle-shaped elements.

8. Pathologically, much light is thrown upon the clinical symptoms, and the great rarity of sarcoma of the heart makes it more important to note the fact that there was never any angina pectoris.

In Dr. Ingram's report of a case called Carcinoma of Heart, in the Transactions of the Philadelphia Pathological Society, 1877, the only case ever presented to this Society, this condition of angina is brought forward as one of the means of diagnosis. In the above case, the report of the Committee on Morbid Growths classes the tumor as an alveolar sarcoma. Secondary sarcomatous growths of the heart are mentioned by a number of authors, but I find no histories giving clinical signs of their existence.

December 14th, 1882.

8. *Specimens showing exuberant vegetations in pericarditis; abundant miliary tubercles of the pleura; tubercles of the liver, of the spleen, of the kidneys, and of the lymph-glands in the region of the head of the pancreas.*

Presented by Dr. SHAKESPEARE.

Dr. Shakespeare could give no detailed history of the case. The subject came from the medical wards of the Philadelphia Hospital, and was last under the care of Dr. James Tyson as visiting physician.

The specimens are brought to the notice of the Society mainly because the autopsy occurred this P.M., and because many of the members have not had opportunities of examining such perfect examples of extensive and diffuse tuberculous infiltration, without more serious involvement of the parenchyma of the lungs.

Examination made six hours after death, of C. J. (colored), aged 60.

Diagnosis.—Pericarditis and pleuritic effusion, with strong bands of adhesions between parietal and visceral pleura.

Thorax.—Left pleural cavity completely obliterated by pleural adhesion. Right pleural cavity contained a large quantity of straw-colored serum. The lobes of this lung were compressed against the spinal column, and were atelectatic. The lower lobe was firmly

adherent to the diaphragm, and the three lobes were strongly united by adhesions. The parietal pleura was thickened, and everywhere studded with minute gray semi-opaque miliary tubercles; the visceral pleura was in a similar condition, except that the tubercles were less numerous. The cut surface of the right lung presented nothing abnormal to the sight save absence of air; but the sense of touch showed, beneath and near to the pleura, a few scattered hard, minute points, much less than millet seeds. The pleura of the left lung was also studded with numerous miliary tubercles, and the tissue of this lung was similar to that of the right. It was, however, crepitant. Pericardial sac contained two and a half ounces of straw-colored serum. The whole heart was covered with an exuberant crop of vegetations. The cardiac walls were perhaps slightly softer than usual. The organ was not otherwise abnormal.

Abdomen.—Peritoneum, both visceral and parietal, normal. No abdominal effusion. Liver slightly enlarged, capsule apparently normal, surface raised here and there by flat elevations, ranging in size from a hemp-seed to that of a hazel-nut. These were firm and yellowish in tint; the intervening tissue was dark-red. A long, deep incision into the depth of the organ showed the cut surface everywhere presenting yellowish, firm masses of the size above indicated, diffused throughout the tissues, which *latter* were firm, and comparatively normal to touch and sight. The spleen, slightly enlarged, was firm, and infiltrated in a manner precisely similar to the liver. The lymph-glands around the head of the pancreas (which was normal), were much enlarged, but not at all softened, and they did not seem caseous. It may be said, at this point, that no caseous focus was discovered anywhere. The kidneys appeared normal to the naked eye, except that there were in each organ one or two yellowish points, having more or less of a pyramidal shape.

None of the joints were examined, neither was the brain.

Several points of interest in this case are worthy of notice. 1st. The family history is not known. 2d. Aspiration was several times performed, and some quantities of pleuritic effusion drawn off. 3d. What was the origin of the numerous tuberculous irruptions, and if there was auto-infection, what was its probable course?

It is to be hoped that Prof. Tyson will give the Society some information concerning the history and course of this case ante-mortem.

Dr. Formad mentioned a case where the enormous thickening of the peritoneum by plastic and tuberculous infiltration gave rise to the

suspicion of malignant disease. He also detailed five cases free from hereditary taint where frequent tapping had induced tuberculosis.

Dr. Tyson said he regretted having to admit that he was less familiar with the history of the case than he should have been, since the patient was in his own wards in the Philadelphia Hospital. The case had been previously thoroughly studied by Dr. Bruen, whose absence he regretted, as Dr. Bruen knew so much more about the case. The man had, however, been more than once tapped. He was a colored sailor, aged 60. When Dr. Tyson took charge of the ward in September, the man presented the physical signs of double pleuritic effusions, orthopnoea, a feeble trammelled heart, but no cardiac murmur. There was œdema of the legs. He was tapped with great benefit, and under a restorative treatment he rapidly improved, so that he soon became one of the walking cases in the ward, attracting little attention. About Dec. 1, he became very much worse. The orthopnoea, and other signs of accumulating fluid returned, and so did the œdema of the legs. His urine was repeatedly examined for albumen, with negative results. He was tapped upon the right side, and three pints of fluid removed, with but partial relief. The other side was also aspirated, without success. A cardiac friction sound was noted, which seemed to be pleuro-pericardial, but in the light of the autopsy it was probably pericardial. He died on the 13th of Dec. 1881.

With reference to the case cited by Dr. Formad, it had also seemed to be a simple plastic pleurisy with feeble distant heart-sounds, without murmur. The case was one where, after prolonged illness, death resulted from exhaustion. Dr. Tyson had never seen in serous inflammations such extensive malformations resulting from the large lymphoid masses in the abdomen, having at first suggested the idea of malignant disease. The patient had a distinctly scrofulous history. The father had died after pleurisy, with cheesy deposits, followed by miliary tuberculosis of the lungs later in life. Four or five uncles and one aunt had all died phthisical from between 20 to 40 years of age.

Dr. Bartholow asked if the range of temperature accorded with that usually found in phthisical cases.

Dr. Tyson said that in the case mentioned by Dr. Formad, the temperature was seldom if ever above 101° , mostly below this point. In the case reported by Dr. Shakespeare, he knew nothing about the temperature.

Dr. Nancrede demurred to the view that repeated tapping had any causative relation to the development of tubercle after pleurisy, but

thought that the chronicity of the affection and altered condition of the pleural sac, which demanded frequent operations, were the real explanation of this fact.

Dr. Tyson said that there had been no head symptoms in the sailor's case. In the case mentioned by Dr. Formad, decided mental aberration, demanding watchful restraint, had been present. No inflammation of the meninges had been found, the only disease consisting of a small tubercle, starting from the pia mater, which dipped down into the brain substance.

Dr. Musser said that a relationship between pleurisy and pulmonary tuberculosis could not be denied, but whether the pleurisy or the tuberculosis be antecedent, was difficult of solution. That the former is primary, may be inferred from the fact that persons are considered as threatened with phthisis who have subclavian arterial murmurs, due to the pressure or pulling on the artery of organized lymph. Likewise various friction sounds and exocardial murmurs are noted to precede tuberculosis, and especially to occur in those tubercularly predisposed. Examples of both classes of cases had come under his observation. It seemed to him that a primary acute plastic pleurisy is a rarity, occurring in a non-tubercular subject. The last series of cases he had seen of what would be called primary pleurisy were in persons predisposed to tubercle, and in some of the cases tubercle subsequently developed. So-called primary plastic pleurisy occurs only in tubercularly inclined individuals; other forms are secondary to some other process, as Bright's disease, a septic process, etc. Trousseau calls attention to latent pleurisies with effusion, as being often an expression of a tubercular diathesis, while, also, a latent pleurisy may occasion development of that diathesis. Two cases illustrating these views had lately come under the speaker's notice.

Dr. Shakespeare, in closing the debate, made some remarks as to the causes of tuberculosis in general and the manner in which it may become diffused through the organism. In this connection he called attention to the absence of any caseous focus for auto-infection, although he readily admitted that it is quite easy to overlook such a point, if it be minute, even after the most careful and painstaking search. He felt that in this and in similar cases all that can be safely said is that the caseous point was not found. Assuming, for illustration, that the point in the kidney might have been the origin of an auto-infection in this case, he referred to the communication between the left renal vein

and the inferior mesenteric vein, and the establishment in this manner of a direct connection with the portal system.

With respect to the point raised by Dr. Musser, he said that he could not doubt the existence of a plastic pleuritis separate and distinct from tuberculosis. He himself had frequently had occasion to examine, under the microscope, sections of lung and pleuritic adhesions, and knew that he had seen very many sections of such adhesions which presented no evidence of tubercle. Moreover, writers who have produced systematic works upon pathological histology and who have made more or less extensive and thorough studies upon man and the lower animals, have distinctly recognized a plastic pleuritis, which is, so to speak, idiopathic, quite as positively as they have described a plastic pleuritis associated with tubercle.

He questioned the causative relation of tapping to tubercle, urged by Dr. Formad. He did not believe that in a patient having no tendency to tuberculosis there was more risk of producing a tubercular irruption by tapping, than, for instance, by puncturing of an anasarctous limb.

He was very well aware of the facts dwelt upon by Dr. Formad and Dr. Musser respectively, viz., the association of tapping with tuberculosis, and the association of plastic pleurisy with tuberculosis, but believed that the frequency of this association has been much exaggerated.

He thought that, in view of the well-grounded belief that in certain classes of animals as well as in certain families of men, inflammations tend to linger, to produce accumulations which are prone to degeneration and to *excite* local or general tuberculosis, it is more logical to conclude that, in such cases as above mentioned, there is at the outstart a tainted constitution—a soil already sown with the dormant seeds of disease waiting to be warmed and awakened to their active powers of destruction by the stimulation of an *exciting cause*. The more frequent the action of the exciting cause the more certain is this dormant tendency to be aroused.

He commented upon the fact that two cases of the five which Dr. Formad had brought to the notice of the Society as examples wherein tuberculosis had occurred in individuals free from hereditary taint, could be shown not to belong to that category. Dr. Shakespeare cautioned the Society against accepting as a genuine instance of tuberculosis, without hereditary predisposition, any case whatever in which the most rigid and complete inquiries into antecedents and collaterals for at least three generations have not been made. Two of the most certainly hereditary of all hereditary affections are retinitis pigmentosa

(night-blindness) and color blindness. Yet it is well known that these diseases often skip whole generations—appearing only in the grandchildren, and even then only in some, whilst others of the same generation escape.

Hence, the records should be scanned with the most careful scrutiny. Especially is this caution required with the middle and lower classes everywhere, and with all classes in America, where it may be said to be the custom to be ignorant even of the names of grandparents and their brothers and sisters, much more of the diseases with which they were afflicted.

Is any one sanguine enough to hope to meet such rigid requirements in a place like Blockley Almshouse, or even among the patients of the best hospitals in the land? How many cases, even in private practice, can the oldest and most experienced physician point to in which a complete history for three generations has been obtained? And yet, unless we have this complete record in thousands of cases, it seems unwarrantable to deny the hereditary tendency of tuberculosis, and extremely injudicious to magnify an exciting into a producing cause.

December 28th, 1882.

9. *The morphology of pulmonary phthisis.*

Read by Dr. EDWARD T. BRUEN.

The etiology of phthisis is very properly exciting careful attention at present; but the subject is in far too unsettled a condition to permit even a useful discussion. I shall not, therefore, allude to it.

A consideration of special interest seems to me to be connected with the morphology of primary tubercle. In certain individuals, owing to inherited tendency, or particularly unfavorable surroundings, recovery after bronchitis due to cold is retarded, or a susceptibility to a new bronchitis is increased. In either case, lingering catarrh, in the majority of instances, is the inception of the series of pathological processes known as phthisis pulmonalis. Two microscopical appearances nearly identical occur, but they differ materially in their microscopic anatomy. Certain so-called miliary tubercles are frequently composed only of the inflammatory products of connective tissue, without the characteristic true tubercular arrangement. The word miliary expresses their appearance, but the inflammatory products may be so arranged as to represent true tubercle, pseudo-tubercle, or

diffuse inflammation. This tubercle, or pseudo-tubercle, is constant in the different forms of phthisis in the adult, except in the instance of pure interstitial pneumonia. Frequently the amount of tubercle tissue in the lungs is so great as to form the principal part of the process, although complicating inflammatory cheesy products are also present.

CLASSIFICATION OF PHTHISIS.

Tubercular peri-bronchitis is probably the best term for the earliest stage of phthisis, and sometimes is the best to designate the process all the way through. The appearance of the peri-bronchial tissues resembles berries on a stalk. The formation extends along the bronchi, spreading from acinus to acinus until the trunk is reached, and is also distributed in the sheaths of the vessels and lymphatics. True tubercle may penetrate a bronchus and involve the lining membrane, and a true tuberculous ulcer may form the basis of an extensive associated bronchitis. Aside from this, these infiltrations excite interlobular connective-tissue growth, and subsequently the walls of the vesicles become thickened, and some vesicular catarrh ensues, which may occlude a lobule. Thus the three divisions of the pulmonary tissue share in the pathological process of early phthisis.

Desquamative pneumonia.—The cause of more than nineteen-twentieths of vesicular consolidation is a result of a process of desquamative pneumonia. This term is used to describe the diffuse inflammation which may accompany the former processes, and which more than the other changes paves the way for the disintegration of the lung. In this process the peripheral epithelial cells of the bronchi are proliferated and shed, thus filling the bronchi and infundibula, while the surrounding connective tissue becomes infiltrated with cells. In children this process is a common one, on account of the greater cellular activity in these subjects. In the desquamative catarrhal pneumonias of early life, the process differs from the desquamative pneumonias of adults in that the air-cells only are filled, and there is little or no change in the intervesicular tissue. Hence it is that recovery is so much more frequent in children than in adults. This process of desquamative catarrh is the basis of that acutely developed phthisis which follows croupous or catarrhal pneumonia, and which has been called acute catarrhal phthisis or galloping consumption—sometimes pneumonic phthisis. When the pathological process thus described is less rapid, the result which follows has been classed

by some as acute caseous pneumonia. When the changes are still more slowly developed, it is synonymous with the chronic catarrhal pneumonia. The relative development then of these processes known as phthisis, in the three divisions of the pulmonary tissues, the bronchial, the interlobular, and the vesicular, is dominated by the activity of the inflammatory process—peri-bronchitis, with consecutive changes, in the latent forms, desquamative pneumonia in the more acute forms; while a process presently to be alluded to—interstitial phthisis—occurs as a very slowly developed change. In many of these cases, when the destruction of the lung is very rapid, the tubercular deposition, true or pseudo, occurs in the early stages, but is masked in the later by the development of the secondary inflammatory desquamative pneumonic processes.

The situation at which phthisis is developed is probably most frequently the apex. The forces of expiration presumably are less efficient at this side, and the lungs are less entirely freed from mucus. The circulation also is less than at the roots, and the products of inflammation are therefore dryer. The roots of the lungs, however, in a large majority of cases are primarily affected. This is especially true of cases in which the original processes of invasion are latent.

I pause here to observe a clinical point of some interest, viz., that when the area of the lung involved in the process of phthisis is distinctly limited, and does not shade off gradually into healthy lung, my opinion is that a favorable result may be very possible. Certainly I have seen the process of phthisis arrested, even where it had passed into the stage of cavity, when the lesions were sharply defined. The localization of a lesion, other things being equal, is a point of favorable prognosis.

Another topic of interest is the share taken by pleurisy in the development of phthisis. A specimen already exhibited before the Society is again shown to-night. It is taken from a colored man, seventy years old, with a family history free from the taint of pulmonary disease. He was tapped five times for the relief of hydrothorax due to heart failure. Each paracentesis was followed by an exacerbation of pleurisy. Nine months after the first operation death occurred from an increase of the hydrothorax. The autopsy disclosed general miliary tuberculosis of the pleura and secondary deposits in the viscera; throughout the parenchyma of the lung, adjacent to the pleura, there was a copious deposit of tubercle, but the other parts of the lungs were normal. Another specimen exhibits the same tendency, viz.,

general pleurisy with phthisis and cheesy deposit in the pulmonary parenchyma, with cavities. The deposits are most abundant near the pleural surface of the lung, the deeper parts evidencing a more recent date of formation. These specimens show that pleurisy may give origin to a tuberculous inflammation. But while this is true, it is conceded by all that dry pleurisy is a frequent secondary lesion in the progress of pulmonary tuberculosis. Pleurisy is, however, in many cases very conservative, since by the thickening and adhesion of the pleural surfaces the ulceration of the walls of superficial cavities is arrested and pneumothorax is prevented.

Interstitial phthisis or *cirrhosis* is a process in which true or pseudo tubercle may or may not be associated. The pulmonary tissues are traversed by narrow bands of connective tissue which may gradually compress it more and more, finally converting it into dense fibrous masses. The color of the lung is apt to be slaty and dark. However, in some cases of interstitial phthisis, patches of the peri-bronchial and desquamative pneumonia may be found, with cheesy degeneration. Interstitial formation is an important part of the process by which cavities are inclosed and sometimes cicatrized.

Distribution.—Interstitial phthisis, if consecutive to bronchitis, is usually bilateral, affecting the upper lobes, although as a unilateral affection it is not infrequent.

Syphilitic phthisis.—It is of interest to note in this connection that the early lesions of syphilitic phthisis so-called are evoked very often by bronchial catarrhal inflammation, which predisposes to structural changes. In these cases there is a principal interstitial fibro-nuclear growth, commencing in the alveolar wall and concentrically arranged around the smallest bronchi and pulmonary vessels. Wagner maintains that the alveolar wall is implicated as commonly in syphilis as in ordinary phthisis.

Bronchial narrowing occurs in these cases by the pressure of the new growth which develops along their lumen. Bronchial occlusion may occur from this new formation, but it is also caused by the enlarged bronchial glands, one of the effects of syphilis. By this means serious mischief in the lungs may be developed, in kind proportioned to the degree of obstruction, such as atelectasis, emphysema, and certain forms of pneumonia. Green and Virchow suggest that the origin of syphilitic diseases of the lung is distinctive in this respect, that while in ordinary phthisis the fibroid is secondary or co-equal in its development with changes in the alveoli and alveolar wall, in syphilis

there are primarily, and chiefly, interstitial changes. Later, when entire vesicular consolidation and breaking-down occur, the process is similar to ordinary phthisis and indistinguishable from it. The vascularity of the new growth of connective tissue is also claimed to be a distinctive characteristic of the inflammatory proliferation due to syphilis. But we must remember, in any discussion of early syphilitic lung disease, that the one special and characteristic lesion of syphilis is the change in the intima of the bloodvessels. This has not yet been demonstrated in the lung, but merely general thickening of the external coat of the vessels. In the effect of interstitial processes upon the bronchial tubes the syphilitic differs from the non-specific disease. The tough, contracting, fibrous tissue which radiates through the lung draws together the bronchial tubes, and deforms by narrowing or flattening them, possibly even to obliteration. On the contrary, in the forms of non-syphilitic fibroid phthisis the bronchial tubes are widened. The process proceeds, in syphilis, from the hilus into the interior of the lung, following the tract of the bronchial radicals and the bronchial arteries. The lesions also occur on the surface, near the visceral pleura, where there is also more connective tissue. Gummata occur as a later process. Time will not allow me to allude to these as I should like.

Miliary tuberculosis.—A demonstration of the pathology of phthisis would be incomplete without including some cases of true miliary tuberculosis. This process may be primary in the lungs, or secondary, as a part of the general infiltration of the serous and mucous membranes, the lymphatic glands and the viscera. This form of tubercle is characteristically recognizable only in the miliary stage. Its appearance as a number of small, hard, translucent nodules is too familiar to need description. A specimen upon the table illustrates an extensive lymphangitis of the pulmonary pleura, forming a network over the pleura.

The process microscopically shows adenoid tissue in nodular form. Similar cases were described before the London Pathological Society in 1880.

Enlargement of the bronchial glands.—Another important part of the phthisical process is the enlargement of the bronchial glands. They present a firm, pigmented character, and the connective tissue is usually infiltrated. I have observed in many cases in individuals suffering from temporary catarrhal conditions of the bronchial mucous membrane, especially when there is a family history of inherited phthisis, but particularly in distinctly scrofulous persons, a set of symptoms referable to enlargement of these glands. These symptoms consist chiefly in an

alteration of the rhythm of the breathing, presumably from pneumogastric irritation; inability to fill the chest with air and a sense of suffocation are complained of; added to these there is *pain* in the back to the right or left of the second dorsal vertebra. Further details of the clinical *ensemble* would carry one away from the pathology of the subject. When the enlargement of the bronchial glands is excessive, it may occasion severe mediastinal pressure, and pain becomes an important clinical symptom, and is of the sort occasioned by mediastinal growths generally. To detect this enlargement during life, Geneau de Mussy has suggested percussion over the spinous processes of the cervical vertebræ in the course of the trachea. Following this line, in healthy subjects a distinct tubular sound is elicited by percussion down to the point of bifurcation of the trachea at the level of the third dorsal vertebra.

Opposite the fifth and downwards we get a lower pitched sound, due to the pulmonary resonance. When the tracheal or bronchial glands are enlarged, the tubular sound over the upper dorsal vertebræ is replaced by dulness, which may contrast sharply above with the tracheal and below with the vesicular resonance. The result of bronchial pressure upon the pulmonary tissues is best marked when the processes of phthisis are not too extensive; also in interstitial phthisis, or in cases where there are marked bronchitic complications. In these cases the lumina of the bronchi are seriously diminished, and vesicular air supply is interfered with. Consequently, emphysema with or without asthma, atelectasis, or a very intractable bronchitis, may occur.

I will not describe the morbid process of phthisis in detail. The involvement of an entire lung is simply the filling up of the parenchyma with peri-bronchial product, or with the results of desquamative pneumonic or interstitial processes. And as one or the other of these predominates, so do we have peri-bronchial, fibroid, or catarrhal phthisis.

Another interesting although not demonstrable incident in the pathology of phthisis is hemorrhage. Bleeding from the lungs occurs both early and late in the history of cases. The late hemorrhage is easy to explain, being nearly always due to the ulceration of the bloodvessel walls. The cause of early hemorrhage is less simple; it is possible that in cases of phthisis there may be malnutrition or fatty degeneration of the bloodvessel walls, rendering rupture under conditions of increased arterial tension in the lungs an easy circumstance. It may also be the result of tubercular infiltration of the muscular walls, which is followed by rupture of the bloodvessels. Cavities in phthisis are

the result of several processes. They occur (a) by a slow or rapid process of fatty degeneration, followed by ulceration; (b) as the result of chronic bronchitis and softening of bronchial tissue, with subsequent yielding to traction from without; for instance, in bronchial pneumonia or fibroid phthisis; (c) from abscesses, as a sequence of acute lobar pneumonia, following hepatization or purulent infiltration; (d) as the direct result of gangrene, itself the immediate consequence of wounds of the lung or blood poisoning, or of emboli. Local gangrene on a small scale occurs sometimes around cavities in lungs or in the bronchial tubes, and may give rise to temporary fetor of breath, but is not likely to lead to fallacious inferences, chiefly because of its temporary character and the absence of permanent concomitant symptoms. It naturally follows that there are two locations for vomicae, the pulmonary and bronchial tissues. Specimens illustrating the various forms of cavities are upon the table.

The limits of a paper designed to open a discussion on phthisis will not permit me to dwell upon the bearing of these pathological changes upon physical diagnosis. I therefore will close with a brief *résumé* of the general clinical symptoms which define the diagnosis of the various sorts of cavities.

Phthisical cavities commonly are situated in one or both lungs, and are indicated as a development in a train of symptoms which include as prominent features gradual emaciation, persistent loss of weight by reason of non-assimilation of food, more or less frequent hemorrhage, and hectic, frequent pulse, hacking intermittent cough, nummular sputa, expectorated in varying amounts throughout the twenty-four hours, and not periodically as in bronchial dilatations, nor inaugurated by a gush of pus and mucus, as in abscess.

Cavities of the nature of abscesses.—The pathology of these cavities, with their coincident clinical history, is not that of phthisis. The history of these cavities is either recovery by contraction (especially after wounds), or more frequently the abscess grows larger and larger until the entire lung may be destroyed, in this respect resembling phthisical cavities. Where death occurs, it is by exhaustion and hectic; where recovery takes place, it is by free opening externally or internally and evacuation of the contents. At times, the small amount of constitutional disturbance, slight degree of emaciation, good pulse, easy breathing, slight cough, and healthy complexion, are in noticeable contrast with the physical signs. Cavities of the nature just described are mostly located in the bases of the lungs.

Cavities due to bronchial dilatations.—Frequently for years the general health is almost unimpaired, and it is never so proportionately to the degree indicated by the physical signs. There are neither hemorrhages nor night-sweats, and emaciation is not a pronounced symptom. The same physical signs persist for months or years unchanged, contrary to the history of most phthysical cavities, which continually alter with the advancing malady. The expectoration of bronchial dilatation is more abundant, fluid and purulent than in catarrhal phthisis, and is usually brought up in the morning or evening by the cupful. It is not a constant spitting of nummular sputa, as in true consumption. In chronic cases the expectoration may become so fetid as to generate suspicions of gangrene. The cough is harassing, but is often relieved if the bronchial cavity is thoroughly emptied.

Dr. Musser said that little could be added to this admirable and concise, yet exhaustive *résumé* of Dr. Bruen on the morphology of phthisis. The limited experience he had had in the study of the histology of phthisis had convinced him that in the microscopical structure of the lesions there is but little difference noteworthy in the primary changes. We have one school teaching that inflammation, another that tubercle, is the primary element. I cannot but agree with the author of the paper in the statement that the disease varies histologically with the variance in the intensity of one of these elements. In both we have epithelial proliferation and accumulation, changes in the vascular and lymphatic tissues (tubercle), and increase in the interlobular connective tissue. So intimately is the evolution of each of these processes connected, that a classification like that of Green (*Lancet*, '82) seems most proper: A. Consolidation, intra-alveolar. B. Consolidation, involving mainly alveolar walls. C. Consolidation, consisting largely of interlobular connective tissue. Without absolute committal either way, and yet in the line of exact truth, the distinction thus given seems to cover the entire histological ground. Dr. Musser has had abundant opportunity for the study of the clinical aspect of phthisis, and although an arbitrary histological distinction of its varieties cannot be made, it is of the utmost importance as influencing treatment, and hence the prognosis of the case, to have a distinct and definite idea of the clinical varieties. The importance of the subject will be sufficient apology for the clinical remarks. Ante- and post-mortem observation has led him to adopt the following classification as convenient, systematic, and embodying the various phases of the disease.

- A. Acute.
 - 1. Catarrhal pneumonia.
 - 2. Pneumonic or caseous phthisis.
 - 3. Pulmonary tuberculosis.
 - 4. Miliary tuberculosis.
- B. Chronic.
 - 1. Catarrhal pneumonia.
 - Tubercle may be secondary.
 - 2. Tuberculosis.
 - 3. Interstitial or Fibroid Phthisis.
 - Tubercle may be secondary.

It is scarcely fair or proper to call acute catarrhal pneumonia a kind of phthisis, as it only is related as a possible primary factor. So seldom is it recognized, and so baneful are the results of non-recognition, and the consequent inactive and inadequate treatment, that it is important to show its relation. It will be observed that catarrh and tubercle are distinguished, and so for convenience and contrast we may term the kinds "catarrhal" and "tubercular."

Perhaps a clue to the pathology of phthisis may be found in defining the characteristics of each. In the first place, there is a marked difference in the predisposing causes of the disease—hereditary and diathetic condition. Thus tubercular phthisis is markedly hereditary; catarrhal is not; the tubercular is associated with the tubercular diathesis and phthinoid chest, the catarrhal in some with the scrofulous diathesis and a perfect chest. Then the mode of onset differs greatly. In the former, the general symptoms are more marked, the pulmonary symptoms in abeyance; in the latter, the pulmonary symptoms are more marked, the general slight. Emaciation, loss of appetite, and dyspepsia precede or accompany the development of the former; they do not occur until late in the latter. Amenorrhœa and changes in the voice also occur early in tubercular phthisis. In tubercular phthisis hemorrhages occur more frequently and earlier than in catarrhal. In tubercular phthisis dyspnœa is a more marked and early symptom, and is out of proportion to the physical signs. Debility is more marked and more readily induced in tubercular phthisis. The temperature range is not so high early, and does not have the long daily sweeps in the late stages in tubercular as in catarrhal phthisis. With a "doubtful mark" it seems pleurisy and chest-pain are more common and constant in the tubercular than in the catarrhal form. The physical signs are bilateral, and not pronounced in the tubercular form; they are unilateral, and pronounced in the catarrhal form. The progress of the tubercular form is rapidly and progressively

downward; of the catarrhal, slow, and in spurts. Tubercular phthisis is contagious (?) and auto-infective. Over and over again do cases present themselves at the University Hospital and Dispensary with just such definite and broad distinctions, which distinctions should determine the line of treatment to be adopted, the catarrhal form requiring a more active local and general antiphlogistic treatment. It is not to be forgotten that the cases are not always, I may say, generally, so easily distinguished, while the picture does not apply to acute miliary tuberculosis or fibroid phthisis.

In a previous discussion on phthisis Dr. Musser had held that acute primary plastic pleurisy did not occur, save in a tubercular subject, and hence was secondary to that diathesis. That statement is possibly too broad, and it should be that frequently-recurring acute pleurisies occur only in the tubercularly diathetic, and are antecedent to the development of phthisis. The recognition of exocardial and subclavian murmurs leads one to say that persons having such sounds are *threatened* with phthisis.

Dr. Eskridge said that he had been much interested in Dr. Bruen's remarks, in most of which he concurred. As to pleurisy antedating phthisis, it was an old view, which had lately been revived. He had been struck with the frequency of chest-pains preceding phthisis; in over 700 cases of which he had records, this symptom was noted in more than two-thirds. This pain may be on the side opposite to that of the affected lung. Interstitial phthisis is not always a chronic affection, Dr. Barlow and others having reported cases which proved fatal in six months. Hemorrhage in his experience was a very common symptom in this variety of phthisis. Dr. Eskridge called attention to the fact that cardiac valvular diseases, especially mitral regurgitation, attended by venous congestions and coming on after birth, are rarely associated with phthisis, while congenital deformities of the heart, attended by venous stasis, etc., are followed, according to some eminent authorities, in nine-tenths of the cases, by tuberculosis of the lungs.

Dr. Shakespeare said that one point made by the lecturer had especially struck him as being opposed to his own experience, viz., that the earliest part of the lungs attacked was generally the root. He had usually found the consolidation at the periphery of the lobe, often forming, as it were, a shell of solid lung, perhaps an inch thick, while the central or deep portions would be either not at all, or but little affected. This peripheral consolidation seemed to have no particular relation to initial pleurisies, for the consolidations were quite frequently

met with in cases almost free from pleurisy and old adhesions. He had also very often noticed at autopsies, after death from intercurrent disease, wedge-shaped patches of solidified lung tissue having their bases at the periphery of the organ and a slightly raised surface, much like infarcts and containing miliary tubercle, whilst the intervening pulmonary tissue was normal.

Dr. Formad asked Dr. Bruen what he considered was the difference in the pathology of acute and chronic phthisis; also on which side the disease most commonly occurred. Dr. Formad also desired to put on record some new observations on the histology of phthisis, made by W. H. Mercur in the Pathological Laboratory in the University of Pennsylvania, which he narrated as follows: That acute phthisis (all fatal cases) is invariably complicated with croupous pneumonia, which conditions the lethal termination; that the lining of the bronchioles and the endothelium of the bloodvessels play a very active part in the formation of organized tubercle granulations, filling and widely distending the lumina in both instances. The existing observations on this point, as far as I remember, refer only to bloodvessels, and then merely to cheesy, broken-down material obliterating vessels, or occasionally to the formation of giant-cells, or to something which in transverse section simulates a giant-cell. Mr. Mercur states that the obliteration of bronchioles by living, organized granulation tissue, is the most common starting-point for pulmonary tubercle granulations, and forms the greater bulk of the latter. He also found that the exudate within the air-vesicles in acute phthisis was capable of undergoing complete organization, and that a group of such blocked-up air-vesicles with organized exudate is usually called (erroneously) a miliary tubercle, the outlines of the air-vesicles being mistaken for submiliary tubercles. Mr. Mercur has failed to find a single true miliary tubercle in a large number of thoroughly studied cases of phthisis, and agrees with those who regard miliary tubercle-nodes as secondary products only. Dr. Formad desired distinctly to state that Mr. Mercur's observations were made on, and apply only to, the lungs.

Dr. Tyson was interested to note how much histological investigation has contributed to our knowledge of the nature of the important processes now under discussion; and while he was ready to admit that we owe much to experimental pathology he felt that our present more correct notions were the result of microscopic studies of the human tubercular lung. The point to be insisted upon is, that all these processes are tubercular, and all are inflammatory, the catarrh of the

lung and tubercle granulations being in all cases the initial lesion, whence it extends peripherally by desquamative catarrhal pneumonia, or centripetally by a tuberculous peri-bronchitis, the former furnishing the rapid and the latter the slow forms of phthisis. That pleurisy is often the initial lesion of tuberculosis he thought had long been acknowledged.

Dr. Shakespeare said that the observations reported by Dr. Formad for his pupil reflected great credit upon both, and were another testimony of the value of the work done in the pathological laboratory of the University, but at least two of the announcements, for which novelty and originality were claimed, had been forestalled years ago by other observers. He had particular reference to the organization of the products within the alveoli of the lung, and the announcement that the walls of the minute bloodvessels, by a proliferating endoarteritis and periarteritis, formed the miliary tubercle. The former is not only recognized and described in Green's Handbook of Pathology, but is also most beautifully *illustrated*. The latter has been repeatedly observed and published, sometimes with illustrations. This origin of tubercle is distinctly referred to in the text-books of Wagner, and Cornil and Ranvier. All these books are in the hands of the University students.

Dr. Bruen, in closing the debate upon the subject, said that he coincided with Dr. Tyson in the opinion that phthisis was rarely associated with heart disease. In mitral regurgitation there was often, indeed, a thickening of the pulmonary substance, allied to the inductive changes in the other organs from like causes. Advanced fibroid disease with cavities, as shown in one of the specimens exhibited by him through the kindness of Dr. Hinsdale, he had not met with heretofore. Dr. Formad's query as to the relative frequency of phthisis upon the right or left side, he felt must be answered by the statement that probably, in a small majority, the right side was most frequently involved. He thought that the peripheral portions of the apices anteriorly were the most common starting-points, where there was much desquamative pneumonia and rapid phthisis, while the roots posteriorly were primarily attacked in the more slowly developed forms of broncho-pneumonic phthisis. The roots of the lungs are the seats of the latent phthisis, developed as the sequential lesion of croupous pneumonia. Dr. Bruen dissented from the view that croupous pneumonia was a frequent cause of death in phthisis. He believed that

in the rapid, as well as in the more latent form of phthisis, death was preceded and hastened by a development of the tubercular nodular tissue to which allusion had been already made. This tissue completely fills up and chokes the acinous pulmonary structure, causing dyspnœa, etc. etc.

February 8th, 1883.

10. *Metastatic pneumonia following a pistol-shot wound of the temporal bone.*

Presented by Dr. H. M. FISHER.

The patient, H. G., aged 26, from whom the specimens I present to-night were taken, was admitted to Dr. Levis's wards, at the Pennsylvania Hospital, October 31, 1882, suffering from a pistol-shot wound of the right ear. The following clinical notes of the case were kindly furnished to me by Dr. J. H. Wills, resident surgeon to the hospital.

Examination of the wound with the Nélaton probe showed that the ball had passed through the posterior wall of the external auditory canal, and was firmly imbedded in the bone about one inch from its point of entrance. The seat of the ball was distinctly located by the marking of the lead on the porcelain of the Nélaton probe.

A prolonged effort to extract the ball was deemed inadvisable on account of its close proximity to the lateral sinus. On account of the inflammatory œdema of the tissues around the wound, poultices were applied. Ptosis of the right lid and slight depression of the right corner of the mouth were noticed.

The patient remained in the hospital four weeks, during which time his general health appeared fairly good. He was permitted to go out on a pass, returned the following day with well-marked pleuro-pneumonia, and died ten days later.

The autopsy showed that the ball had penetrated the tympanic cavity and passed into the jugular fossa, where it was found imbedded. There was marked periostitis in the neighborhood of the wound, but neither inflammation of the walls of the lateral sinus, nor of the internal jugular vein, was noticed.

I am inclined to believe that the infarcts found in the lungs and pleuræ owed their origin to numerous small thrombi that had been formed in the diploë of the petrous portion of the temporal bone, the

venules of the diploë having become plugged as a result of the periosteal inflammation. These thrombi, becoming loosened in consequence of increased blood pressure, were washed out into some one of the adjacent sinuses, and so into the pulmonary venous circulation. The case is, I think, an interesting one, as showing the dangers attendant upon such injuries to the temporal bone, apart from any incidental injury to the brain or its membranes.

Sections of the heart, liver, spleen, and kidneys showed marked amyloid infiltration of these organs. Whether such marked changes could be induced in all these organs in the short period that elapsed from the receipt of the injury to the date of the patient's death is a question I would leave to other members of the Society to decide. At any rate, as far as I could ascertain, no previous history of suppuration was elicited from the patient during the time of his sojourn in the hospital.

Appended are the notes of the microscopical examination of the different organs.

Lungs.—Vessels everywhere greatly dilated, and present numerous infarctions; the alveolar capillaries are also greatly distended, and their endothelium is swollen. Intra-alveolar spaces packed with lymphoid cells and embryonic connective-tissue cells. The alveoli contain accumulations of epithelioid, highly pigmented cells. These cells are generally found associated with exudative corpuscles in a delicate reticulum. These catarrhal intra-alveolar accumulations show in places a tendency to become organized.

The *pleura* is greatly thickened, its vessels dilated and infarcted.

Heart.—Muscular fibres show commencing fatty degeneration, and there is marked amyloid infiltration of the walls of its smaller nutrient vessels.

Liver.—Cell-nuclei alone take carmine staining. Islands of liver-cells found in a translucent, nearly homogeneous basement tissue. Evident amyloid infiltration of the walls of the finer hepatic arteries and biliary ducts.

Kidney.—The Malpighian corpuscles show stiffened capillary loops and little shrinkage from the action of the alcohol. The lumina of the arterioles are patulous. Homogeneous, translucent spaces are seen between the smooth muscular fibres of these vessels.

Spleen.—The lymphoid corpuscles of the pulp appeared to me to present an unduly homogeneous, translucent appearance. The smaller vessels show distinct amyloid infiltration of their walls. The Mal-

pighian bodies also show a notable infiltration, having, in some cases, been converted into amorphous masses, which are semi-transparent, and show interspersed among them a few connective-tissue nuclei.

March 8th, 1883.

11. *Specimens (lungs, heart, and kidney) from a case of Bright's disease.*

Presented by Dr. FREDERICK P. HENRY.

The organs upon the table were taken from a typical case of chronic parenchymatous nephritis. The kidneys are slightly enlarged, extremely pale, with marked arborescent vascularity of the surface, and with non-adherent capsules. In fact, they represent one of those instances in which the gross appearance suffices for diagnosis. The heart is slightly enlarged, its left ventricle dilated, its walls fatty, and its valves competent. The heart gave rise to no murmur strictly so called, but did produce in a typical degree the sound called by Bouilland and Potain the *bruit-de-galop*. Potain considers this sound to be diagnostic of interstitial nephritis, but, for my part, I have invariably encountered it in cases of Bright's disease in which the parenchymatous affection was predominant. The lungs are congested and œdematous throughout. The pleura is greatly thickened on both sides, and the pleural cavities abolished, with the exception of the diaphragmatic portion of the right side, which contains a small amount of fluid in the shape of a number of serum-containing loculi. The cause of death was œdema of the lung, and the interesting question arises as to whether œdema of the lungs, which is so common an event in cases of Bright's disease, may not be favored by the obliteration of the pleural cavity also commonly present in these cases; or, in other words, and conversely, whether the pleural cavity may not serve as an outlet for effusions which, but for its presence, would infiltrate the lung parenchyma. Although my attention has not been specially directed to the point which I have just raised, and, therefore, I have no cases other than that just reported to bring forward in its support, I think I can state, in general terms, that before œdema of the lungs occurs, in cases with open pleural cavity, that cavity contains a considerable quantity of fluid; that is to say, that its capacity to act as a drain to the lung tissue is exhausted, and it does not at all follow that, for this result to

be produced, the pleural sac should be *distended* with fluid. In fact it would seem that a moderate amount of fluid in the pleural sac may be sufficient to exhaust this conservative power, and this amount varies with the state of the pulmonary circulation. The view just advanced is in complete harmony with what we know of the physiological function of lymph spaces, among which the large serous sacs are reckoned. The last volume of our Transactions¹ contains a paper upon pleural effusions, in which I argued at considerable length that any cause which interfered with a proper expansion of the thorax would lead to congestion of the pulmonary capillaries, thereby favoring pleural effusion. The results of capillary congestion vary in different systemic conditions. In Bright's disease, owing to the altered state of the blood, the result is œdema, that is, a serous infiltration of the lymph spaces. Effusion into the serous investment of organs such as the lungs and heart, is conservative as far as concerns the parenchyma of these organs. The practical result of these considerations is that œdema of the lungs in cases of Bright's disease is favored by pleuritic adhesions; and, secondly, that œdema of the lung may perhaps be averted by the timely performance of paracentesis.

May 10th, 1883.

¹ Volume X.

V. THE GENITO-URINARY APPARATUS.

a. KIDNEYS AND BLADDER.

1. *Horseshoe kidney.*

Presented by Dr. H. F. FORMAD.

Dr. Formad said that he simply showed the specimen on account of its rarity, as the organ was, although of abnormal form, healthy. The connecting bond between the two lateral halves was also unusually thick.

Dr. Shakespeare said that this was the second specimen which had been shown before the Society during the past year. Both specimens were removed from patients dying at Blockley.

December 8th, 1881.

2. *Tumor of the suprarenal body.*

Presented by Dr. H. F. FORMAD.

Dr. Formad said that he had no history of this specimen, it having been accidentally discovered in a post-mortem examination made for another affection in a patient dying in the Philadelphia Hospital under the care of Dr. Tyson. The kidney of the same side was of the large white variety, but was unconnected with the tumor, which was developed within the suprarenal body. It had extended also beyond its envelope, and, the doctor thought, probably involved some of the neighboring nerve plexuses. Microscopically it resembled a glioma, but on more careful examination it might prove to be a non-medullated neuroma. There also seemed to be some myxomatous degeneration.

Dr. Tyson recalled the autopsy, but nothing of the previous history of the case. He did not think that the growth sprang from the suprarenal body.

January 26th, 1882.

3. *Case of papilloma of the bladder.*

Presented by Dr. H. F. FORMAD.

Dr. Formad said that this case, while pursuing only the ordinary course of such a disease, and thus being presented without detailed history, was of great interest, since the diagnosis of its nature was made by the microscope. The sediment of the urine when placed beneath the microscope showed many little tips of mucoid tissue, coated with a delicate cylindrical epithelium, which were evidently the terminal points of the dendritic growths.

Dr. Tyson said that he had now a case under observation of suspected papilloma of the bladder, where repeated microscopical examinations had failed to reveal anything characteristic, although the rational signs, such as hemorrhage, etc., clearly indicated some such neoplasm. He had made the diagnosis of papilloma of the rectum, however, by the microscopical examination of the dejecta.

Dr. Hazlehurst recalled the account of a case published in the *Archives of Medicine*, where the microscope had determined the diagnosis.

Dr. Nancrede made some remarks on the close resemblance, as far as microscopic appearances went, of the so-called adenoid and carcinomatous growths of the rectum, and pointed out that, as Dr. Van Buren has shown, the more sessile a growth is the more likely is it to prove malignant irrespective of its histological structure, and, *vice versa*, the same arrangement of cells, stroma, etc., in a markedly pedunculated growth clinically means benignity.

Dr. Tyson said that the history of this case, as well as others seen by him, proves most clearly what has been repeatedly adverted to in this Society, viz.,—that, howsoever malignant an internal growth may be, it does not produce *cachexia*, except when involving some one of the vital organs concerned in *nutrition*, unless, of course, inducing this condition by repeated hemorrhages or profuse discharges.

February 9th, 1882.

4. *Calcification of the bladder.*

Presented by Dr. H. F. FORMAD.

Dr. Formad said that Dr. Dixon had asked him to present this specimen for him without history, since the condition was only acci-

dently found during the post-mortem examination of a man who died of phthisis. No bladder-symptoms had at any time been noticed, yet it seemed almost impossible that an organ with apparently a completely calcareously degenerated lining membrane could have performed its functions so well as not to have had any attention called to its functional insufficiency.

February 9th, 1882.

5. *Hypertrophy of the prostate gland, accompanied by profuse and fatal hemorrhage.*

Presented by Dr. J. B. ROBERTS, for Drs. J. M. ADLER and WM. HUNT.

The clinical history furnished by Dr. Adler is as follows: The patient, aged 66 years, of medium size, weighing 150 pounds, of regular habits, had enjoyed good health until within one year past. On a number of occasions during the past year he has had slight hæmaturia. He passed his urine with ease, and only complained of slight perineal pain. On the 12th of September last he arose in the morning, in his usual health, but soon after breakfast he was attacked with sudden, acute pain in the bladder, which he was unable to empty. Dr. Adler saw him in a short time, when the patient complained of great hypogastric pain, and was much prostrated, with a blanched, sallow, cold skin, and a rapid and feeble pulse; a well-defined pyriform swelling occupied the hypogastric region, extending upward to the umbilicus. After stimulants and morphia had been given, about one pint of fluid blood was drawn off by the catheter, after which, this instrument becoming blocked, a double one was introduced, by means of which injections of warm water were thrown into the bladder, and another pint of broken-down coagula were removed. A solution of alum, 20 grains to the pint, was then introduced into the bladder, and allowed to remain. Despite the internal use of *ol. terebinth.* and *ol. erigeron*, with opium suppositories, the hemorrhage continued, necessitating recourse to the catheter, injections, etc., to free the bladder from coagula. Death ensued on the sixth day, from exhaustion induced by the repeated hemorrhages. The diagnosis arrived at by Drs. Hunt and Adler was carcinoma of the neck of the bladder.

Sectio cadaveris.—The autopsy was a partial one, made in the undertaking establishment. Upon incising the hypogastrium, the distended bladder was at once seen, containing nearly a pint of clotted

blood. This was removed by the hand, through an opening into the viscus, when Dr. Roberts felt, near the vesical neck, protruding into its interior, a pear-shaped mass, about the size and shape of the adult uterus. This, as the members of the Society saw, was evidently a greatly enlarged middle lobe of the prostate gland, covered by unaltered mucous membrane; the lateral lobes were also enlarged. The mucous lining of the viscus was smooth and congested, presenting at one point two small circular depressions, with cleanly cut edges. Owing to circumstances, no further examination of the body was made.

Dr. Roberts said that, as to the probable source of hemorrhage, he would call attention to the two small erosions of the mucous membrane of the bladder.

Report of the Committee on Morbid Growths.—"The specimen presented by Dr. Roberts, upon microscopic examination, is found to consist of the histological elements composing the prostate gland. There is no evidence of any neoplasm, except a numerical hypertrophy of the structures of the organ. The specimen is an hypertrophic prostate gland."
October 26th, 1822.

6. Caseous degeneration of the kidneys.

Presented by Dr. J. B. ROBERTS, for Dr. DUNMIRE.

Owing to Dr. Dunmire not having seen the woman until within a few hours of death, the history is of necessity imperfect. She was a married woman, 39 years old, whose husband is said to have infected her with some form of venereal disease. The husband had been dead for about one year when the patient first came under treatment. There were no evidences of syphilis, so that the supposed venereal affection of the past had been presumably gonorrhœa. When seen September 14, 1882, she was exceedingly ill, and gave a history of general ill health for the past few years, but dated the present trouble some weeks back, when she had bathed in the surf while menstruating. This was followed by a chill, since when she had steadily grown worse. When Dr. Dunmire saw her she complained of sore throat, difficult deglutition, anorexia, sick stomach, pain in the back, with sharp pain running toward the groin, especially on the right side, tenderness over the abdomen, and frequent, scanty micturition. The pulse was 140, the temperature 103°, and there was profuse leucorrhœa. An unfa-

vorable prognosis was given, which was soon verified by her becoming unconscious, and dying six hours later.

Autopsy.—The abdomen alone was allowed to be examined. All organs healthy, except kidneys and bladder. The latter contained a little urine and mucus, its walls were much thickened, and its lining membrane was congested. The left kidney had little true kidney structure left, but was converted into a group of seven or eight cysts, containing a white, cheesy material, of a moderately firm consistence. The ureter was much dilated and thickened for about three inches from the pelvis of the kidney. The right kidney was normal in outline, but when incised revealed one large cyst, with creamy contents, and also another small cavity containing a few minute calculi. Several of these could be felt through the walls of the normal ureter, thus accounting for the ante-mortem renal colic. The perinephritic structures were unchanged, as well as the capsules of the kidneys, although these latter were perhaps more adherent than normal.

October 26th, 1882.

7. *Chronic parenchymatous nephritis, complicating phthisis pulmonalis.*

Presented by Dr. JAS. TYSON.

My object in showing these kidneys is to illustrate the morbid anatomy of the renal complications which so frequently attend the later stages of phthisis pulmonalis. It is very well understood that when œdema of the feet and legs is present in cases of consumption, the end is not far distant, but the renal complication which is at the bottom of such œdema is often overlooked. It is, of course, not impossible that there should be œdema in the last stages of phthisis, from simple alteration in the composition of the blood, a watery state of it, but in the majority of instances it means that the kidneys have become involved. As to the form of disease affecting the kidneys, it is acknowledged that it may be either lardaceous disease or chronic parenchymatous nephritis, but I think the impression prevails—it was at least my own until recently—that the amyloid kidney is the most frequent complication. I believe, however, that chronic parenchymatous nephritis is more common, and it becomes a matter of interest, if not of importance, to be able to diagnose between these

two conditions. It is well known that the microscopic and clinical characters of the urine in these two forms of kidney disease are often identical, so that no assistance is afforded by a study of the urine. The history of the case, of course, leads to neither particular form, but suggests both. One criterion only can I recall to aid us, and that is the presence of an enlarged liver. So commonly associated is the enlarged amyloid liver with amyloid kidney, that the absence of it almost necessarily precludes the presence of amyloid kidney. At least, I am sure we would err less frequently if we were to consider all cases of renal disease attending consumption, unattended by enlarged liver, to be parenchymatous nephritis rather than lardaceous disease. It is true we often have enlarged fatty liver in consumption, but the degree of enlargement never reaches that of the amyloid liver, and hereafter I shall be inclined to consider all cases of renal disease complicating consumption to be parenchymatous nephritis, unless they are associated with enlarged liver, when I shall conclude that they are instances of amyloid disease.

November 9th, 1882.

8. *Sarcoma of the prostate gland.*

Presented by Dr. W. E. HUGHES.

This specimen was taken from a patient in the University Hospital, under the care of Dr. H. R. Wharton.

W. C., aged 35 years, was admitted to the University Hospital suffering from retention of urine. Before his admission numerous unsuccessful attempts had been made to empty the bladder by means of a catheter. On admission the patient complained of much pain in the hypogastric region, which was the seat of a smooth, rounded swelling, reaching almost to the umbilicus. He stated that he had gonorrhœa some years previously, which had been followed by a troublesome stricture, which had been perfectly relieved by the passage of bougies. For more than a year previous to his admission he had suffered at irregular intervals from difficulty in urination. The urine had never been bloody, but its passage had often been attended by great pain. It was found impossible, on account of numerous false passages, to introduce a catheter into the bladder. On introducing a finger into the rectum the prostate was felt smooth, rounded, and immensely enlarged. The patient was put to bed, ordered suppositories of belladonna and opium, and to

have a warm poultice applied to the abdomen. This treatment relieved him almost immediately, and urination became freer. For a few days he did well, but the difficulty in urination soon began to increase, and by the fifth day after his admission the symptoms had become so urgent that it was deemed advisable to repeat the attempt to pass the catheter. This attempt was as futile as the first. Then aspiration of the bladder through the abdominal walls was attempted, but only a small quantity of blood was obtained. In introducing the canula it gave the sensation of passing into a solid body, and careful palpation revealed the fact that there really was a solid body apparently occupying the whole bladder. It was now decided to open the urethra at the base of the bladder, through the perineum, and the operation known as Cock's was selected. The operation was followed by the escape of a small amount of urine. After this the patient did well, with the exception of an attack of dysentery, until the ninth day after the operation, when peritonitis suddenly developed. He died on the following day.

Autopsy two hours after death.—Upon opening the abdomen, a thick, yellowish-red, purulent liquid, having a urinous odor, was found, bathing the intestines, the walls of which were everywhere bound together by recent adhesions. The omentum was in places firmly adherent to the intestines, and contained numerous, irregular, nodulated masses, varying in size from that of a pea to that of a hen's egg. These masses, on section, presented a whitish-yellow color. In the lower part of the abdominal cavity was a large, irregularly shaped tumor, firmly adherent to the small intestines, colon, omentum, and walls of the pelvis. On careful dissection the tumor was found to originate in the prostate gland. On section it apparently presented, in parts, the characteristics of scirrhus, in others, those of encephaloid. No trace of normal prostate gland, nor seminal vesicles could be discovered. The bladder, containing a few ounces of urine, was found in front of the growth, its upper boundary almost on a line with the umbilicus. Its anterior wall was apparently perfectly normal; its posterior wall, resting on the tumor, was thickened, raised, red, and velvety. The ureters were normal, and opened in the usual position. The urethra, as far as could be seen, ran along the anterior surface of the tumor, and was not involved by it. The weight of the growth was five pounds two ounces. The kidneys, stomach, lungs, and intestines were normal. The peritoneum and capsules of the liver and spleen contained several secondary growths. The brain was not examined. Microscopic examination

showed the growth to be a typical, small round-celled sarcoma. The secondary deposits were similar in structure to the primary growth. The submucous and muscular tissues of the bladder wall were somewhat infiltrated. The growths in the capsules of the liver and spleen had commenced to penetrate those organs. *November 23d, 1882.*

9. *Case of vesical calculus.*

Presented by Dr. C. M. WILSON.

This specimen, which I am privileged to show you, is a uric acid calculus. It has the characteristic appearance of this variety of calculus, and is chiefly remarkable for its size, which is fully that of a large hen's egg. Its weight before some fragments were clipped off for chemical analysis was 1140 grains. The following is the history of the case:—

A. E., æt. 20, white, a coal-miner, had complained of vesical pain and difficult micturition for the past four years. The symptoms steadily became worse during that time, and have become especially aggravated during the last five months. The prominent symptoms, upon his admission into the hospital, were intense pain in the bladder, and the incontinence of retention, the stone falling down into and occupying the bas-fond of the bladder, thus occluding the urethral orifice. The patient was admitted on the afternoon of September 1, 1882. An examination by Dr. Levis revealed the existence of the stone. From that time until the morning of the 3d, when Dr. Levis performed the operation of median lithotomy, he was kept moderately under the influence of opium, and the bladder was evacuated every four hours with a soft catheter. Dr. Levis operated on the morning of the 3d, and removed the stone, making a very large wound into the bladder. *April 12th, 1883.*

10. *Phosphatic vesical calculi.*

Presented by Dr. C. M. WILSON.

These specimens were taken from a man 72 years of age, by Prof. S. D. Gross, and are examples of phosphatic calculi. They are thirteen in number. The triple phosphates have been deposited over many centres, instead of being aggregated into one, as in the previous specimen. Their combined weight was 1200 grains. The patient made a good recovery. *April 12th, 1883.*

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11. *Sarcoma of the bladder.*

Presented by Dr. J. H. MUSSER.

I am indebted to my friend Dr. Samuel R. Skillern, for whom I made the autopsy, for the privilege of exhibiting this very interesting specimen. At the time of the examination the skin of the body was of the characteristic cachectic hue, the rigor mortis was marked, the emaciation considerable, though not striking, for on section there was a fair amount of fat in the abdominal walls, and in the omentum, while the muscles were comparatively large. The abdominal cavity alone was examined. The peritoneum was healthy, the stomach and intestines of a normal appearance. Neither the mesenteric nor any other lymphatic glands were affected. The liver was slightly enlarged and fatty; the spleen normal. The genito-urinary tract was removed intact, the kidneys being severed from their attachments with difficulty, on account of being surrounded by fat. Beginning with the kidneys, the left was about half the natural size, with thickened adherent capsule. The pelvis was very greatly dilated, the secreting portion reduced to one-third the natural size. The medullary portion mostly atrophied, the cortical was thin, hard, and pale. The right kidney was larger by one-third than normal; it was also cirrhotic, and, although its pelvis was dilated, the secreting portion was not atrophied very much. The ureters were very much dilated, averaging the size of the index finger. The bladder was in its normal position, and on opening, its capacity was found lessened by one-half; the walls were much hypertrophied. At the base of the bladder, in the trigone vesicale, a flat tumor was detected. It measured two inches transversely, and one inch antero-posteriorly. The base of the tumor was smaller than the upper surface. This surface was irregular, at some places ulcerated, at others covered with phosphatic concretion. The orifices of the ureters were found by hydrostatic means to open into the tumor, and hence were somewhat occluded. The urethral canal was not encroached upon. To the left of the large tumor were two small secondary masses.

Microscopical examination of the kidneys and the tumor.—In the former, interstitial and tubular nephritis was found. The tumor was of the histological appearance of a sarcoma. The sections are under the microscope for examination.

The person from whom these specimens were removed had been a

successful minister, in charge of a large congregation. He had always been a great mental worker. At the time of his death he was fifty years old. During his life his habits were most exemplary. His previous health was good; in his family history no evidence of hereditary disease could be traced. For the clinical history I am indebted to the various medical gentlemen that attended him.

Prof. Agnew was consulted more than two years prior to death, on account of vesical irritation. Six months thereafter he passed blood by the urethra. Careful examination at this time and before, both by the urethra and rectum, failed to detect any tumor or calculus. In a short time the cachectic hue became evident, and with all these symptoms the Professor suspected malignant disease. The hemorrhages became more profuse, and occurred more frequently. Prof. Tyson was then consulted. He very kindly allowed me to extract the following from his notes.

Dr. Tyson did not see him again until October 31, 1881, when he reported that during the previous year he had been using the bougie at one time as often as five or six times a week, but more commonly every three or four days. He had been very well during this time, gaining ten pounds in his summer vacation, and at that time not urinating more frequently than any one else, although he kept up the use of Poland water, of which he was using eight tumblerfuls a day for five weeks while at the spring and when at home five a day. He now mentioned that during the summer he felt an occasional soreness in the region of the bladder when stooping, and noticed also occasional chalky deposits in his urine. At the time of this visit his urine was acid; sp. gr. 1014, and contained one-fifth its bulk of albumen, but no tube-casts. No note of blood was then made.

On November 7, his urine contained a sediment equal to about one-tenth to one-twelfth its bulk, which was composed mainly of blood corpuscles. There were a few leucocytes. He continued to visit Dr. Tyson until March 28, 1882. During much of this time he reported himself improved, there being, at times, much less frequent urination while the uncomfortable sensation at the neck of the bladder was less. The Poland water was discontinued and ordinary drinking water substituted, with about the same effect in relieving the symptoms. During most of this time he took oil of eucalyptus in doses of from six to ten drops three times a day; we thought at the time with good effect. But the same changes occurred when he was taking nothing. His urine was always albuminous, sometimes containing blood appreciable

to the naked eye. Once or twice he passed a small clot of blood from the bladder, and on January 7, 1882, a large clot which he compared to a small leech. Two or three times he brought chalky concretions, evidently phosphates, which he had passed. He always thought the bougie relieved him. He was always worse after a hard day's work, as on a Monday after preaching twice on Sunday. Benzoic acid and ergot were used with about the same effect as the previous remedies. The oil of eucalyptus apparently was of temporary but not of permanent advantage.

On March 28 he reported that since his previous visit, seven weeks before, he had been very ill, with what seemed to be an attack of great prostration, during which the urine was little altered except that there was increased phosphatic sediment. More recently, however, there seemed to be always more or less blood in the urine except for short intervals. At no time during his attendance did he present a cachectic appearance, nor any other symptoms except those mentioned of intermittent hemorrhage from the bladder and symptoms of vesical irritation, and Dr. Tyson had not suspected malignant disease of the bladder while he was under his observation, but thought, rather, there might be a hemorrhoidal condition of the prostatic plexus of vessels.

During the summer and fall of 1882 his general health failed very much, while the vesical irritation was quite pronounced. When under Prof. Agnew's care, he began to use a sound and at this time used it daily for its soothing effect. Along with the slight vesical tenesmus, he suffered from a little pain at the head of the penis from the very first. During this time and in the winter the hemorrhages continued. The blood was discharged before the urine; sometimes a considerable amount of pure blood, fluid or in clots. The bloody discharges sometimes occurred with every urination, or days would pass by with clear discharges.

Dr. Skillern attended him from January 9, 1883, until his death, March 13. From his notes, in addition to the above, I glean the following facts: The occasion of the first visit to the patient was due to a fit which he had had and the nature of which was not clear, although it was probably a syncopal attack from blood loss. He suddenly became unconscious, and when seen had a pallid face, cold, clammy extremities, a feeble pulse and shallow respirations. There had been no convulsion, although slight convulsive movements were noticeable. Hypodermic injections of amyl nitrite soon aroused him, although he dozed for an hour afterwards. There is no evidence to prove that this

seizure was uræmic. At this time his general condition was very bad. During January it is noted that he used the catheter frequently on account of a slight difficulty in starting the flow of urine, and that in using it a grating sensation was felt by him; that the paroxysmal hemorrhages continued; that the constant feeling of discomfort at the neck of the bladder, and the dysuria grew worse. Micturition occurred every two hours. In addition to the above, in February he had morning nausea and vomiting, generally losing his early meal, this accident becoming more frequent later in the month. The loss of flesh and strength became very evident, as did the cachectic appearance. Two weeks prior to death he began to complain of renal pain. For twenty-four hours prior to death the pain was agonizing. He died of exhaustion. It may be noted that the urine was never suppressed, nor was there ever marked vesical pain. Dr. Formad expressed the opinion, from an examination of the urine several weeks before death, that malignant disease of the bladder was present, confirming the opinion of the other gentlemen. His opinion was based on the character of the epithelium in the deposit—this having the appearance of that of the deep layers of the bladder mucous membrane.

Dr. Agnew kindly informs me, without referring to his notes, that he distinctly recalls four cases of sarcoma of the bladder, all in males, and in all bloody urine was an early and constantly recurring symptom, amounting in one of the cases to very profuse hemorrhages. Pain in one or both hips was also an early symptom. The patients were all over forty years of age.

Dr. Tyson said that Mr. S. first came under his observation May 27, 1880, being then 47 years old. He stated that he had been annoyed by frequent micturition for about five years, which gradually increased, until at that time he had to rise two or three times each night, but thought the vesical irritability was more frequent during the day than at night. At first he was completely relieved of this symptom during his vacations, which he spent in the woods, and was still much better at such times. He thought Poland water gave him relief, and he felt compelled to use it constantly. There was at this time a burning sensation at the neck of the bladder. The urine at this date contained one-twelfth its bulk of albumen, enough blood corpuscles to give it a "smoke hue," but no casts. On June 22 he again reported, Prof. Agnew, in the mean time, having passed a sound, and detected a slight stricture, which he thought accounted for the symptoms. The patient was instructed to pass a sound for himself and he thought it gave him great

relief, not only diminishing greatly the desire to pass water, but also relieving the uneasy sensation at the neck of the bladder.

This interesting note calls attention to the chief facts of these cases, and an especially noteworthy one—the age of the patient. As is well known, sarcoma in other situations generally occurs in early life, in this in later life. An examination of the relation of the mouth of the urethra to the flat tumor will show how readily a sound could have passed over the tumor; while the base of the bladder being filled the sound could not engage the mass. The enlarged prostate was no doubt deceptive, obscuring the basal mass, when the rectal touch was resorted to.

May 24th, 1883.

12. *Kidneys and heart from a case of chronic Bright's disease; extreme uræmic dyspnœa; marked œdema; relief to both by acupuncture; incipient optic neuritis.*

Presented by J. H. MUSSER, M.D.

R. T., æt. 35, admitted to the University Hospital July 17, 1878. Single; a packer of goods, frequently exposed to draughts while in profuse perspiration; used tobacco to excess; once or twice a year he would spree. At twenty-two he had a chancre (?); no secondary symptoms. Three times he had gonorrhœa. Always healthy prior to present illness; never had rheumatism.

The patient inherited a tendency to phthisis from both parents, and to rheumatism from his maternal grandmother.

The onset of the present illness was initiated two years ago by a sudden night attack of dyspnœa. The dyspnœa continued for nine weeks, worse at night, and preventing work in the daytime. Œdema of the feet, and frequent micturition accompanied the dyspnœa. He improved, but had a relapse in four weeks, of a month's duration, followed again by temporary improvement, and a third relapse. From the latter he never rallied, œdema, cough, dyspnœa, frequent micturition, and dyspeptic symptoms being constantly present. The cough was dry, and attended by sub-sternal pain. The œdema was general. When admitted to the hospital, under the care of Prof. Pepper, his condition was as above mentioned. During the July, August, and September following, the asthmatic attacks continued. In October they were relieved, but the anasarca became more pronounced.

It may not be out of place to say that the dyspnœa was very severe, and almost defied relief. It presented the clinical character of uræmic asthma, and was relieved only by inhalations of nitrite of amyl, or by hypodermic injections of morphia. The anasarca was very great, and not relieved by diuretics, diaphoretics, or cathartics. In October, acupuncture was resorted to, with temporary relief to the lower extremities and scrotum, its good effect lasting five days only, but being so marked as to encourage one in its use. During the following three months the punctures were made about fifteen times, and after each operation about three pints of serum would drain away. The latter part of December erysipelatous inflammation developed about the punctures on the *right leg*, and extended over it. During the progress of the inflammation, large bullæ would form, the bursting and continued free discharge of which caused an entire disappearance of the œdema of *both legs*. Relief was not only afforded by these operations to the œdema, but the attacks of asthma notably diminished in frequency and severity. January 8, two weeks after the inflammation of the right leg developed, sloughing took place. The sloughing was confined to the calf of the leg, was deep, and was attended with free *serous* discharge from the ulcers. In a few days a typhoid state set in; he rapidly lost strength, and on the 7th of February died of exhaustion.

When these notes were taken (Jan. 8), the remaining features of the case were as follows: He was emaciated and anæmic, and his skin was harsh and dry, his countenance anxious. Appetite poor; flatulent dyspepsia marked; the bowels constipated; hemorrhoids; tympanitic abdomen; slightly enlarged liver; normal spleen.

At the apices of the lungs diminished expansion; flattening, impaired resonance, and increased fremitus at the left; moist crackling, and sub-crepitant rales heard throughout the lungs. Apex beat of heart in sixth interspace, one-fourth inch inside of nipple line. Veins of right side of neck enlarged. Cardiac impulse moderately strong. At apex a low-pitched systolic murmur; muscular element lessened. At base, pulmonary sounds accentuated.

The width of the cardiac area of dulness was increased one-half inch to the left, and was not changed by full inspiration.

Urine contained albumen; amount varying, at times, two-thirds, then one-third bulk; hyaline and numerous granular casts.

Ophthalmoscopic examination.—Small disks. Myopia. O. D. Disk dirty gray, veins tortuous. No marked change in the color of the

nerve; outlines misty, and the disk is slightly swollen. O. S. Disk same, but more marked. No hemorrhage in either eye. Diagnosis, incipient neuritis.

Autopsy five hours after death.—No rigor mortis; body emaciated; commencing ecchymoses; great fulness of the venous circulation. Lungs: apices bound down by adhesions. Slight right hydrothorax. Base of left lung, anteriorly and posteriorly, adherent. At left apex three or four areas of catarrhal pneumonia. Heart: weight twenty-one ounces; left ventricular wall hypertrophied; mitral valve insufficient, admitting almost three fingers; its cusps thickened; one leaflet of the aortic valve slightly diseased. Left cavities increased in size. Aorta atheromatous, a large patch is seen, one-half inch from the valves. Liver enlarged, hard, congested. Kidneys small, congested, capsules adherent, relation of cortical to medullary substance normal.

Microscopical examination.—Kidney showed decided interstitial nephritis, with fatty degeneration of the tubular epithelium. Liver slightly cirrhotic and fatty. Muscular fibres of the heart had undergone slight fatty degeneration.

June 14th, 1883.

13. *A specimen of renal calculus; oxalate of lime.*

Presented by Dr. J. H. MUSSER.

A. R., æt. 32, a stone-cutter, residing one year in Philadelphia, applied to me for treatment June 5, bringing with him a sample of his urine. It was bloody, and had been voided without pain, during the twenty-four hours prior to this visit. Three months ago, without any cause, he was seized with pain in the left loin. The pain increased in severity, kept him from work, caused faintness, but no nausea or vomiting, did not radiate in any direction, save transversely to a slight extent, lasted three days, and was not followed by hemorrhage. In the intervening three months he was in good health. A jar of the body or any movement did not increase this pain. It may be noted, but is rather irrelevant, that for five or more years he has always had a weak back, becoming painful when stooping.

Twelve hours after the consultation, the same localized pain recurred, and continued for twenty-four hours. Suddenly it was relieved, and the subsequent urinary discharges were clear. In three days he had a return of the pain and hemorrhage simultaneously. The pain

increased in severity, and at the height of the paroxysm extended to the testicles and the head of the penis. The paroxysm was relieved by the passage of the calculus I show you by the urethra. The passage of the stone through the ureter was characterized by the most agonizing pain and frequent attempts to urinate.

Microscopical examination of the first sample of urine showed that the red color was due to blood. A similar examination of his clear urine, as well as the bloody, proved the presence of urate of soda and crystals of the phosphates. The urine was acid. The diagnosis of renal calculus was not difficult, but the microscopical appearances of the urine led me to infer that the stone was composed of uric acid. With such an idea the patient was put on alkaline treatment. The sequel proved that the effect of such treatment was of no avail, save from the diuretic action of the drugs. Morphia was given to relieve pain.

A portion only of the stone was exhibited, an angular portion having been broken off by the patient. The portion weighs .06 gram. The exterior is tuberculated; the interior dark in color, bluish, the exterior lamina light brown. It is exceedingly hard. Chemical examination, by Dr. Leffmann, showed it to be an oxalate of lime calculus.

June 28th, 1883.

b. FEMALE ORGANS OF GENERATION.

14. *Ruptured ovarian cyst ; death from peritonitis.*

Presented by Dr. M. LONGSTRETH for Dr. J. G. LEE.

Emma Curnell, white, female, æt. 34 years, height 5 feet 2 inches, born in London, married some sixteen years, died November 1, 1881, at 5.20 P. M. Had no children, except one fifteen years ago. Always menstruated regularly, and was in tolerably good health, except that she complained occasionally of a pain in her right side.

Two months ago she began menstruating, and continued passing blood from the womb for somewhat over five weeks, which caused her to go to one of the dispensaries in this city. What was done for her relief there I was unable to ascertain; but on October 25 she was obliged to take to her bed, with severe pains in the abdomen, and was seen on October 29 by Dr. James Simpson, who found her "with a pulse of 84, skin cold, and covered with a cold, clammy sweat, bowels

very loose, vomiting incessantly, and she was constantly tossing herself from side to side of the bed, moaning with pain. Soreness on pressure over the abdomen; no tympany." Dr. Simpson was told that she had been in the same condition for five days, nothing having been done for her relief. Dr. Simpson directed turpentine stupes to the abdomen, morphia for the relief of pain, and that after she was relieved of the pain she was to take a pill of acetate of lead and a small quantity of opium, to check the looseness of the bowels. The Doctor called on the 31st of October, and found her apparently much better. Vomiting checked; bowels stopped; belly soft, but tender; no tympanitic condition. He directed tinct. cinchona and carefully restricted diet. He was again called in November 1, in the early part of the afternoon, and "found her pulseless, cold, but partially conscious; pupils normal, responding to light, but she was evidently suffering excruciating pain in the lower part of the abdomen," and moaned when he made the lightest pressure anywhere about the abdomen; "the belly was tympanitic." The family told him she had been suffering all the morning from pain. She died two hours later.

Post-mortem twenty-one hours after death.—Body in ice. Rigor mortis partially developed. No external marks of violence. Purging from mouth. Abdomen considerably distended. No marks of violence on genitals. Upper portion of vagina containing some sanguineous matter. Peritoneal cavity contained about a pint of yellowish liquid. Omentum attached to intestines and bladder by recent inflammatory adhesions. Intestines firmly matted together by fresh inflammatory adhesions. Peritoneal walls, convex surface of liver, and under surface of diaphragm covered with freshly-effused lymph. The uterus was about the ordinary size of a multiparous organ, and its anterior surface seemed to be normal. On the right side, behind the broad ligament, extending from the pelvic wall to about the middle of the posterior surface of the uterus, was a cyst about three inches in diameter, tightly adherent to the uterus. On separating the margins of the adhesions, they appeared as if cut with a knife. The greater portion of the cyst-wall is sufficiently thin to be translucent, while the portions about the ovary are firm, rigid, and at places one-half inch in thickness. Nearly the whole of the peritoneal surface is covered with inflammatory lymph, also the uterus, ovaries, etc. The lining of the cyst is smooth. Lying at the junction of the cyst and the uterus is an oval body, apparently continuous with the cyst, which on section showed several small openings resembling enlarged ovarian follicles. No very

distinct trace of the Fallopian tube could be made out, though a rounded cord-like body passed from the upper corner of the uterus along the upper surface of the cyst, turning to reach the position of the ovary. The ovary and Fallopian tube of the left side are firmly matted together by old inflammatory adhesions. The os uteri is dilated; the neck and cavity of the cervix were ulcerated or inflamed. The uterine cavity appears normal. No probe can be passed into right Fallopian tube.

Heart, lungs, liver, kidneys, spleen, and other organs healthy. Head was not opened.

Cause of death.—Peritonitis (shock), the result of a rupture of an ovarian cyst.
December 8th, 1881.

15. *Cancerous degeneration of a fibro-myoma of the uterus, with metastatic growths in the brain, etc.*

Presented by Dr. J. M. BARTON.

Mrs. R., æt. 42 years, mother of three children, the youngest now fifteen years of age, first noticed a tumor in the right iliac fossa ten years ago. In 1877, when she first came under my care, she had an enlarged abdomen, full of fluid. After tapping, on September 17, 1877, and drawing off thirty-four pints of straw-colored fluid, I found an irregular mass firmly bound down in the right iliac fossa, reaching to the median line and as high as the umbilicus. It was connected with the uterus, into which I was able to pass a sound to the depth of five and one-half inches, and subsequently to the depth of nine and one-half inches. Before tapping, the intestines did not float upon the surface of the fluid, but were bound to the posterior wall of the abdomen, and could be felt as a doughy mass after the fluid was removed. The diagnosis was fibroid tumor of the uterus, while the fluid and position of the intestines were regarded as due to a coexisting chronic peritonitis. Each menstrual epoch was prolonged, and the blood lost was much in excess of the normal amount; on several occasions it was so profuse as to necessitate tamponing of the vagina. The fluid rapidly reaccumulated, and I again tapped her on January 12, April 12, July 17, and November 16, 1878, and on July 8, 1879, and again on January 15, 1882, removing each time, except the last, from twelve to eighteen quarts of fluid.

During 1878 several small cysts ruptured into the abdominal cavity. The patient, while attending to some domestic duties, would have severe pain in the abdomen, fainting, feeble, and rapid pulse, pallor of countenance, and other evidences of loss of blood. After remaining in bed for several days, her strength would return, and at the next tapping the fluid would be mixed with dark grumous blood.

While taking ergot steadily, the uterine tumor decreased in size, the hemorrhages ceased, the general health improved, and the fluid accumulated much less rapidly. After the tapping in 1879, she enjoyed good health, and was able to attend actively to her household duties, until the latter part of 1881, when she had an offensive discharge from the vagina, rapid pulse, loss of appetite, etc. She improved under treatment, but, as the distended abdomen interfered with respiration, I tapped her in January with a large aspirator needle, three and a half inches to the left of the umbilicus, drawing off only three quarts of dark, bloody fluid. It was then found that the solid growth had much increased, and was two inches greater in each diameter than ever before. Above the solid growth was a large fluctuating, circumscribed mass.

About the first of the year three small, rapidly-growing, semi-solid tumors appeared, one on the forehead, one on the right forearm, and one on the left arm below the axilla.

About the 1st of February, though still quite strong enough to walk about the house, the patient's mind became suddenly affected; there were some delusions; her memory could not be relied on even regarding events occurring but a few minutes before. She would frequently fail to understand the simplest question, and would repeat her unmeaning reply again and again. On the 12th paralysis of the right arm occurred, on the 15th she became comatose, and on the 16th she died.

Post-mortem.—The uterine tumor, besides its normal attachments, had fastened itself to a small portion of the abdominal wall at the umbilicus, and to the omentum. The lymphatics of this portion of the omentum are filled with material resembling the metastatic growths. The posterior lobes of both the right and left halves of the cerebrum have in each a tumor the size of a walnut; the dura mater has also a deposit about an inch in diameter, opposite the middle of the sagittal suture. The skull was affected at the same point, a small nodule attracting attention while separating the scalp. The intestines were firmly bound to the posterior portion of the abdominal walls by old adhesions.

I here present the uterine tumor, the omentum with enlarged lymphatics, the two posterior lobes of the cerebrum, a portion of the dura mater, and one of the superficial tumors.

Dr. S. W. Gross did not think it possible that a fibro-myoma could undergo carcinomatous degeneration. *February 23d, 1882.*

16. *Carcinoma of the bladder and uterus.*

Presented by Dr. J. M. BARTON.

No written history accompanied this case, owing to the patient having unexpectedly expired the night previous. There had been nothing of special interest in the case before death, but the extremely sudden and unexplainable death induced the exhibitor to present the specimens.

Dr. Tyson hazarded the suggestion that in such a chronic case latent kidney trouble might have escaped detection, which the mode of death described by Dr. Barton was somewhat suggestive of.

March 9th, 1882.

17. *Polypi from the uterine cervix.*

Presented by Dr. W. H. PARISH.

Dr. Parish exhibited two small uterine growths, each about the size of the last phalanx of the thumb, removed December 28, with the wire écraseur. On Christmas eve he saw for the first time a patient, of French birth, a teacher, apparently about 35 years old. When he entered her room she was in a state of syncope, from hemorrhage from the genitals. The hemorrhage had, however, ceased. With the application of hot wet cloths over the front of the chest, and hypodermic injections of whiskey and spirits of ammonia, in a few minutes she rallied, so as to be able to tell the Doctor that she had not menstruated for three months, when bleeding suddenly began from the womb, and continued during the day with an exacerbation, just before sending for him. As the patient's condition was evidently a critical one, Dr. Parish asked the direct question if she had not had, or was not having an abortion? She said it was impossible. He then learned that she was single, and 42 years of age. A digital examina-

tion showed a hymen present, and a substance in the vagina that, at first touch, felt very like an embryo of about the third month. Dr. Parish, however, soon recognized that it was a growth attached to the lower part of the cervical canal, and that there was another distending the cervix. The latter felt still more like an embryo or ovum; and, in fact, in the absence of the one in the vagina, it might have at first given rise to the error of thinking that the patient was aborting. Slight traction on the vaginal tumor showed it to be attached.

There was no return of the bleeding, and December 28, with the assistance of Dr. M. O'Hara, Dr. Parish removed both the growths with the wire, without etherization or the use of a speculum.

A marked feature of the patient's history is that she had always menstruated scantily, and often at intervals of five or six weeks. Never before had she evinced a tendency to uterine hemorrhage. Dr. Parish was confident the patient was not pregnant.

Report of the Committee on Morbid Growths.—"A microscopical examination of a section made from the growth removed from the uterus, shows it to be adenomatous in structure, consisting of small cavities or spaces lined with cells, which cavities are separated from one another by fibrillar connective tissue." *December 28th, 1882.*

18. *Condylomatous tumor of the labium.*

Presented by Dr. NANCREDE for Dr. C. M. SELTZER.

Mrs. ——— contracted syphilis nine years ago from her first husband. Has had specific treatment at intervals, but never prolonged beyond a few weeks each time. Condylomata and ulcerations around the vulva, anus, and perineal region were first noticed about three years ago. She first came under my observation and treatment June 1, 1882, at which time symptoms of her disease were very pronounced, such as cachexia, nervousness, nocturnal headache, ulcers on various portions of the body, numerous condylomata, of which the specimen exhibited was the largest, being a cauliflower-like excrescence springing from the left labium majorem, having a pedicle about two and a half inches broad. The clitoris was hypertrophied, forming a knob one inch in diameter. There was urinary incontinence, and consequently severe excoriations of the external genitals. Potass. iod. and hydrarg. bichlorid. were given, upon which there was pronounced systemic improvement. Local

treatment failed to remove the condylomata, hence on the 13th of November two double ligatures were passed through the pedicle of the largest, so as to divide it into three parts. The ligatures were then tied tightly, the mass removed, and the stump cauterized with nitric acid. The clitoris was so sensitive, even under profound anæsthesia, that it was thought best not to remove it. The patient made a good recovery in ten days, including the cure of the urinary incontinence. Her second husband, to whom she had been married about six years, has no evidence of having syphilis. Her only child by her first husband has stricture of the rectum and obstinate fissure of the anus—improvement of which only began upon the recognition of their specific origin, and the consequent line of treatment.

Report of the Committee on Morbid Growths.—"A section of the growth upon microscopical examination shows it to consist of an external covering formed of the histological elements and arrangement as found in the skin; the papillæ were in places much atrophied and flattened and also in some parts elongated. There were no hair follicles, sweat or sebaceous glands in the section. Below the papillæ was seen fibrous tissue, at some points in an active state of proliferation, and numerous bloodvessels. The growth is a fibrous polypus."

January 11th, 1883.

C. MALE ORGANS OF GENERATION.

19. *Chondroma of testis.*

Presented by Dr. H. F. FORMAD.

The patient from whom this tumor was removed was James Fox, æt. 40, white, and born in Ireland. He was admitted into the University Hospital December 19, 1881. Until within the last eighteen months he was healthy. About a year and a half ago he received a blow on the scrotum. The testicle remained tender for some time. One year ago the right testicle began to enlarge. At times there was slight pain, but not enough to interfere with his work.

When removed by Dr. Agnew, a large tumor involving the right testicle and right side of the scrotum, extending to the external ring, was found. It was heavy, free from fluctuation, not translucent, and did not give impulse to touch on coughing. The superficial veins of the scrotum were enlarged.

Report of the Committee on Morbid Growths.—"The tumor removed from the scrotum, upon microscopical examination, is found to be the testicle, in which are developed numerous small nodules consisting of encapsulated cells separated by a hyaline matrix,—cartilage tissue. The tissue surrounding the small cartilaginous nodules is made up of fibrillar connective tissue in a state of active proliferation; its blood-vessels are mostly seen congested with blood. Here and there are seen the seminiferous ducts, the lining cells of which are undergoing granular degeneration. The small tumor connected with the testicle is found to be an enlarged lymphatic gland." *January 26th, 1882.*

20. *Specimens from a case of uræmia following the introduction of the split bougie.*

Presented by Dr. J. M. BARTON.

Mr. H., æt. 32 years; occupation, merchant; has enjoyed good health for a number of years. Had an attack of gonorrhœa three months ago, which rapidly became gleet. He was under the care of different physicians, the last of whom used the bougie as a method of treatment. The solid instrument was used five or six times during the last month, and the split bougie, which is increased in size after introduction (known in this city as the invention of Dr. S. W. Gross), was used about as frequently. It was used for the last time on Saturday, June 3. It was then dilated to its full extent, without meeting with any resistance. On withdrawing the instrument, it was followed by a gush of blood, and each act of urination on both that day and the next was followed by a gush of blood, though the patient was able to, and did, go out, contrary to the advice of his physician. The blood lost during the two days was estimated at about a pint. On Monday morning the patient had a decided chill, followed by fever. On Monday afternoon, when I saw him for the first time, he micturated in my presence; the first four ounces of urine being clear, then one drachm of blood, then another ounce of urine, and then one ounce of blood, during the passage of which there was considerable tenesmus and pain. This was followed, about half an hour afterwards, by a severe chill, lasting an hour. On the following morning he appeared somewhat stupid and heavy, though no morphia had been used for a number of hours. No urine had been passed since six o'clock the previous evening, and on percussing over the bladder there ap-

peared to be little or none in it. During the day the stupor increased, slight twitchings of the tendons appeared, and though active purges, diaphoretics, etc., were used, the uræmic coma increased, and proved fatal at 11.30 Tuesday night, three days after the introduction of the instrument.

The post-mortem, which was made this morning, gave no evidences of disease in any other organ than in the specimens here presented,—viz., the urethra, bladder, and kidneys. The bladder contained about two fluidounces of dark-colored but translucent urine. The mucous membrane of the urethra was ruptured about three inches below the neck of the bladder. The rest of the urethra, as well as the bladder, appeared healthy. The kidneys were both deeply congested, and bled freely on section.

June 8th, 1882.

21. *Epithelioma of the penis.*

Presented by Dr. FERDINAND H. GROSS.

The specimen I have to present is not of any novel interest to most of the members of the Society, but I thought there might be some who would like to examine it.

J., aged 42, laborer, married, born in Ireland. About a year and seven months ago he noticed for the first time a dry whitish scab on the under surface of the penis, near the frænum. He did not apply to a physician until about five months ago, when the scab, which was then somewhat sore, was cauterized, and a cure promised in three months. The part which was cauterized sloughed away, leaving an ulcer which did not heal, but gradually increased in size. From the early part of April last, about two months before I amputated the penis, he began to apply at irregular times to the dispensary of the German Hospital, and was treated for a short time as an out-patient. My attention was first called to the case a week or two later by the resident surgeon. The patient himself ignorantly believed his disease to be of a syphilitic character, and this view appears to have been adopted by the physician whom he consulted. At the time I saw the case, the ulceration had made deep inroads upon the organ, involving the glans penis, prepuce, and integument, and other structures immediately behind. There existed a considerable mass of neoplastic tissue and a fistulous opening into the urethra. The pain was of a darting kind, and the part was especially painful on being touched. I

judged the disease to be of a cancerous nature, and at my request the resident, Dr. L. D. Brose, removed several small portions for microscopical examination. The diagnosis having been confirmed, I had the patient admitted into the house, and amputated the penis on the 10th instant.

The specimen, as here presented, measures four inches in length, and includes about three-fourths of an inch of the organ beyond the visibly diseased structure. In its greatest circumference, just behind the corona glandis, it measures seven inches. Beyond this point several small nodules in the integument are noticed. Except that one gland in the right groin appears very slightly enlarged, which the patient says has been so a long time, nothing is noticeable about the neighboring lymphatics, and they do not appear to be involved.

I am indebted to my resident, Dr. Brose, for the microscopical examination, and append his note:—

“Microscopical appearances.—Squamous epithelioma. The sections examined were made up of large flat cells, containing one or more nuclei, which were very distinctly seen under a power of three hundred diameters. The cells were seen to be arranged in the form of cones, which penetrated to various depths a vascular connective-tissue stroma. In some of the cylinders the epithelium had been arranged concentrically, forming the characteristic ‘pearly bodies.’ The papillæ of the skin were much hypertrophied, and had undergone proliferation inwardly.”

Up to the time of writing this report, ten days after the operation, the patient continues to do well in every respect. *June 22d, 1882.*

d. MAMMARY GLAND.

22. *Adeno-carcinoma of the mamma.*

Presented by Dr. J. M. BARTON.

Mrs. K., a pale and thin but active married lady, 40 years of age, who had never borne children, but who still menstruated quite regularly, had suffered for five months with a tumor of the right breast. It had not been preceded by any injury that she was aware of, nor was there any hereditary carcinomatous taint. The tumor was the seat of sticking pains, particularly at night, and its rate of growth had been quite regular. The opposite breast was much atrophied, and was less than

one-half the size of the diseased one. The disease involved the entire gland; the skin was infiltrated and adherent, and the veins slightly enlarged. The growth was about four by five inches in its diameters and two inches in length, perfectly regular in its outlines, and moderately hard. The nipple was retracted; the mass was freely movable over the pectoral muscle, and there was no glandular involvement. On June 28 I removed the growth, together with some of the surrounding skin. The hemorrhage, though free, was much less than one would have expected from a sarcomatous growth of such size. The large open ulcer healed rapidly without complications, and has been entirely closed for some weeks.

The microscopic section which I have here shows that the increase in size of the breast is mainly due to glandular elements. The ducts, acini, and fibrous stroma are in nearly normal proportions to each other. Many of the ducts have an increased number of, and some are distended with, epithelial cells. Occasionally typical alveoli filled with epithelial cells are found. They are much more numerous in the skin than elsewhere, and the disease might here be regarded as fully developed scirrhus carcinoma.

October 13th, 1881.

23. *Secondary scirrhus carcinoma of the axillary glands.*

Presented by Dr. J. M. BARTON.

Mrs. B., æt. 60 years, a stout married lady, mother of thirteen children, was operated upon in March, 1878, for a carcinoma of the right breast. It had been noticed only for a few weeks, but, as the gland was very voluminous and the growth gave no pain, it had probably existed for a much longer time. I removed at that time but about one-half of the gland, that being fully sufficient to remove all the diseased as well as a large amount of healthy tissue; and as now, forty-one months after, there has been no local recurrence, the operation was probably sufficiently extensive. The tumor was about the size of a hen's egg, was not the seat of pain, and there was no glandular involvement. It had not been immediately preceded by any injury, but many years previously the same gland had been the seat of an abscess, the scars of which still remain even after the removal of the disease, showing, at least, that the malignant growth was not developed at the focus of the previous inflammation. The tumor had all

the microscopic appearances of scirrhus. It was not examined microscopically at the time, and, though I preserved the specimen, I am not able now to identify it. Two years later, in February, 1880, I removed a small axillary growth shortly after it was discovered.

Quite recently, October 2, 1881, I removed the present growth. It had been noticed for about one month, was the seat of great pain, and was deeply situated in the axilla, being nearly in contact with the axillary vessels. The wounds, after all the operations, healed without complication; that made in the last operation was healed in about two weeks.

The section which is now under the microscope of the Society for the observation of the members shows the growth to be a scirrhus carcinoma.

The unusually long interval between the returns is a point worthy of note in this case.

November 10th, 1881.

24. *Sarcomatous carcinoma of the mamma.*

Presented by Dr. J. M. BARTON.

Mrs. B., a patient of Dr. Runkle, of this city, a pale, thin, married lady, 51 years of age, who ceased menstruating at 44, was injured four years ago in the left breast. Six months afterwards she noticed a tumor the size of a hazel-nut, which in thirty-three months, or until January of this year, grew to the size of a walnut. For the last nine months, however, it has been increasing much more rapidly, and has been ulcerated for two months.

On examination, October 1, it was about two and one-half inches in its diameters, and elevated above the surface of the breast to about the same distance. It was a soft, irregularly nodulated, ulcerated, bleeding mass, immediately below the nipple, but not including it, freely movable, and involving but little if any of the mammary gland. It had frequently been the seat of hemorrhages—one very decided one three days prior to operation.

There was no axillary or other lymphatic involvement; there had been no pain at any time; the subcutaneous veins were but slightly enlarged, and the skin was diseased to but a short distance around the growth.

I removed the growth in the usual manner, October 4 of this year.

leaving a large open surface to heal by granulation. Owing to the previous hemorrhages, the patient did not bear the loss of blood well at the time of operation; but she soon rallied, and two weeks after the wound had far advanced towards healing, the patient being able to leave her room.

The microscopic section, which I also present, shows large alveoli filled with epithelial cells surrounded by a very scant stroma composed of spindle-cells. It might, therefore, be classed as an encephaloid carcinoma of the variety designated by Gross as sarcomatous carcinoma.

November 10th, 1881.

25. *Scirrhus carcinoma of the mamma.*

Presented by Dr. J. M. BARTON.

Mrs. C. L., æt. 50 years, is the mother of twelve children, the youngest now eight years old. She still occasionally menstruates. There has been no cancer in the family history as far back as the great-grandparents. One year ago the tumor appeared below and to the outside of the right nipple; it had not been preceded by any injury that the patient was aware of, though she had suffered at her first pregnancy, thirty years ago, with abscesses of both breasts. At the time of operation the breast was much larger than the normal one; the growth, surrounded by a large amount of adipose tissue, appeared the size of the adult fist, and was freely movable over the deeper structures.

The nipple was retracted, and numerous small points of dimpling were noticed in the skin: the skin over the healthy breast also showed some dimpling. The superficial veins, as well as the lymphatics, were enlarged. Eight or ten of the latter, each enlarged to the size of a crow-quill, radiated from the centre of the breast to its margin. I removed the entire mammary gland, as well as a large portion of the surrounding tissue, at the German Hospital, on the 3d of the present month. The incision was carried into the axilla, and an enlarged lymphatic gland, not before discovered, was also removed.

February 23d, 1882.

26. *Scirrhus of the mamma associated with empyema.*

Presented by Dr. E. T. BRUEN.

The specimens which I exhibit were taken from the body of a woman, æt. 40 years, who came under my notice at the Philadelphia Hospital about the 1st of March, 1882.

They comprise a tumor of the mammary gland about the size of a hen's egg, which proved, on microscopic examination, to be scirrhus carcinoma; also a portion of the pleura, showing a secondary deposit of the morbid growth. The portion of pleura is that covering the parietes beneath the attachment of the mammary gland on the left side, from which place the mammary tumor was taken. The remainder of the pleura showed the ordinary evidences of an empyema.

The history of the case exhibits these salient features. In the early part of March the patient came into the house complaining of malaise. Physical examination of the chest revealed a large pleural effusion on the left side. Paracentesis was practised, and about 110 ounces of serous fluid were withdrawn. Subsequently the operation was repeated, and the fluid withdrawn was found to be purulent. A drainage-tube was introduced, but the patient succumbed to an attack of erysipelas, after having been under treatment about two months. During the last three years of life, the patient had periodical discharges of blood through the mammary gland, and afterwards, when the tumor had formed, through a point of ulceration on it. This was supposed to be a vicarious menstruation, since the hemorrhage occurred every twenty-eight days, and during the time of its continuance no menstrual flow appeared per vaginam.

The probable sequence of pathological changes in the pleura has seemed to me—1, a development of a pleurisy through the irritation of the deposit of the morbid growth upon the pleura; 2, empyema following the paracentesis. The pleural inflammation was evidently of low type, since no pain or cough or pulmonary symptoms had warned the patient of intra-thoracic trouble. *May 25th, 1882.*

27. *Enlarged lymphatic glands secondary to carcinoma mammæ.*

Presented by Dr. W. H. PARISH.

I show five lymphatic glands removed to-day from a patient from whom I removed a mammary tumor about nine months ago. The case was then reported to the Obstetrical Society, and is published in *The Medical News* of July 8th. The specimen was referred to a committee, and Dr. Beates made a microscopic examination, and concluded that the growth was an adenoma that had undergone carcinomatous change.

Of the enlarged glands presented to-night, three, each about the size of an almond, were removed from the axilla; a fourth, of smaller size, from just below the clavicle, and the fifth from the side of the neck, about an inch above the clavicle. The patient does not present a cachexia.

December 28th, 1882.

28. *Scirrhus carcinoma of the mamma.*

Presented by Dr. J. H. MUSSER for Dr. R. M. GIRVIN.

In November, 1882, Miss —, a lady in good circumstances, with a hereditary tendency to carcinoma and scrofulosis from the mother, noticed, at the upper portion of her breast, a small, extremely tender lump, the size of a hickory-nut. The nodule increased in size, and on the day of removal, February 17, 1883, was as large as a duck's egg. Neither at that time, nor at any other time, was the nipple either diseased or retracted, or the mamma discolored. The breast had never been injured. The lymphatics were not involved. The general health and nutrition were good.

The operation was performed on the above date by Dr. Girvin, on account of paroxysmal pain. So severe was this symptom, that a slight opium habit had developed. The wound healed nicely. There has been no return of the disease (May 20).

Examination of a section of the hard mass in the gland demonstrated it to be a scirrhus carcinoma.

May 24th, 1883.

29. *Adenomatous growth apparently recurrent; in reality an outlying portion of the mamma not removed at a previous operation.*

Presented by Dr. NANCREDE.

The above title really gives the essential points in the history of a patient aged 23 years, upon whom two operations had been performed for a supposed fibroma (adeno-fibroma?) of the breast, which recurring after partial removal of the breast, the whole organ it was supposed had been then removed by another surgeon. Dr. Nancrede had opposed all operation at first, considering that the breast was really not the seat of anything beyond a local induration, the result of injury. He had removed the third growth which, from its history and microscopic appearances, he was satisfied was the result of the irritating drag of the badly-placed cicatrix on a small portion of breast tissue left at the second operation. The clinical lesson taught by this case was clear, viz., the freest possible removal of mammary growths.

May 24th, 1883.

VI. THE NERVOUS SYSTEM.

1. *The brain of a negro murderer.*

Presented by Dr. CHARLES K. MILLS.

The brain exhibited was from a negro who had committed a murder nearly thirty years ago. He was tried, convicted, and sentenced to death, but the Governor of the State would not sign the warrant for his execution. He was pardoned about five years before his death, subsequent to the appearance of paralysis of the left side. He was a quarrelsome drinking man before the commission of the murder, and a fair representative of the criminal class. The paralysis of the left side of the face, and of the left arm and leg, gradually became more profound.

The right hemisphere of the cerebrum and the left hemisphere of the cerebellum were markedly atrophied. The atrophy was most marked in the motor region of the convolutions, particularly in the ascending convolutions. A hard, brownish-black nodule was found isolated in the superior upper portion of the pons Varolii, to the right of the median line. Both cerebral hemispheres showed a decided excess of fissure and deficiency in gyrus development, with a tendency at numerous points to confluence of fissures. Some remarks were made on the physiological significance of atrophy of the *right* hemisphere of the cerebrum and the *left* hemisphere of the cerebellum, and also on the views of Benedikt that the brains of criminals are of the confluent fissure type. The brain was referred to the Committee on Morbid Growths for microscopical examination, and Dr. Mills expressed his intention of recording the case in detail at some future time.

Dr. F. P. Henry referred to a case of bilateral atrophy of the superior and inferior parietal convolutions reported in the February number of the *Archives of Medicine* by Dr. J. C. Shaw. With the exception of being bilateral, the lesion closely resembled that of the specimen just exhibited. The case presented symptoms of mania,

epileptiform convulsions, and slight paralysis, but its greatest interest consisted in the fact of its corroborating some recent experiments of Ferrier, made with a view to determine the situation of certain sensory centres, particularly those of vision and hearing. For full particulars Dr. Henry referred those interested in the subject to the article in the *Archives*.
February 23d, 1882.

2. *Acute hydrocephalus; acute Bright's disease in infancy; death from œdema of the lungs.*

Presented by Dr. J. H. MUSSER.

The infant, a male 3 months old, from whom the specimens were removed, was brought to my notice as a case of acute hydrocephalus. He had had a fall three weeks previously. After the fourth day succeeding the fall, the mother noticed the head gradually increasing in size, without fever or any evidence of meningeal irritation save the utterance of short cries in the midst of his sleep, occasional vomiting, and a tendency to stupor. He nursed well, and his bowels were regular.

At the time of the visit the head was greatly enlarged, measuring three inches from auditory meatus to meatus, and being nineteen inches in circumference. The scalp was tense, the hair rather thin, the veins enlarged, the fontanels and sutures widely distended, so that fluctuation was manifest. The axes of the eyes looked downward; there was slight internal strabismus. The child was as bright as infants of his age, and was well nourished.

The first visit was made on the 17th of March, 1882. From thence until death no special cerebral symptoms developed. The "cry" and the vomiting continued, the latter but twice daily. On the 24th of April he was brought to me with general anasarca. It had been of three days' duration. The urine was scanty, and it was with difficulty that two drachms were obtained. It was three-fourths albumen, and contained numerous hyaline and pale granular casts. The anasarca increased, and death took place in seven days, from œdema of the lungs, or about nine weeks after the fall.

Autopsy twenty-four hours after death.—Rigor mortis; general œdema. Brain and kidneys only examined. Membranes of the base adherent, thickened, and opaque, with not very recent lymph in the

fissures. Lining membrane of the ventricles granular and thickened; velum matted. The ventricles contained a quart of clear fluid, in which a trace of albumen was detected; no urea or sugar. The substance of the brain was very pale; the convolutions effaced. The kidneys were enlarged and congested. The capsule was easily removed; it gaped when incised. The exterior of the glands were mottled. On section, the cortical portion was much congested and swollen, of a dark-red color, with here and there punctiform hemorrhages. Microscopical examination of the kidneys showed changes which clearly indicated an acute catarrhal nephritis.

The cause of the Bright's disease was either the poison of diphtheria or scarlatina. A sister had the former disease at the time of the baby's death, while the latter disease was prevalent in the "row." The case is of interest on account of the early age of the child. I know of no record of a child so young having had acute Bright's disease.

May 28th, 1882.

3. *Specimens from a man who died from brain, lung, heart, liver, spleen, kidney, and bladder troubles.*

Presented by Dr. J. T. ESKRIDGE.

Charles S., æt. 68 years, born in Germany, a tanner; thirty years ago he came to America, where he almost uninterruptedly followed his occupation until the last few years of his life, when he was disabled by disease. His father died æt. seventy-two years, from some obscure trouble; his mother, æt. seventy-eight, died from supposed heart and kidney diseases. No family history of carcinoma or phthisis was obtainable. So far as I was able to ascertain he had never contracted any venereal disease. Soon after his arrival in this country he suffered from some brain trouble, when he suddenly lost his hearing; but the left ear soon improved, while the right remained deaf. About ten years ago he became subject to attacks of jaundice, his skin gradually becoming permanently discolored.

My attention was first called to him in the year 1877, when I found him jaundiced and quite weak. His lungs were emphysematous, his heart feeble, and beating slowly (fifty to sixty per minute). A mitral systolic murmur, which was thought to be organic, was heard. Incontinence of urine, which began a few years before, was then troubling

him. A dull, heavy, sometimes sharp, shooting pain was complained of in the region of the liver. His jaundiced condition became more intense, his bowels constipated, and he gradually grew weaker as his appetite lessened. During the first two years that he was under my care, his urine was repeatedly examined chemically, but no albumen was detected, notwithstanding the presence of a dull, heavy pain in the back, and a dropsical condition around the ankles and under the eyes. Growing weaker, I advised him to stop work two years ago, and ordered him to take tonics and wine regularly. The last two years of his life he complained of headache and oppression over the heart and upper portion of the chest. The dropsy did not increase, but the pain in the hepatic region became more severe, and his strength gradually failed. During the two weeks preceding his death he was troubled with nausea and pain in the region of the stomach.

October 26, 1882, he retired to bed about 10 P. M. feeling not unusually weak. The next morning he was so prostrated that when he attempted to rise he was unable to assume the erect posture. He complained of a tight feeling over the upper portion of the sternum. His bowels had not been opened for a week, the secretion of urine was less than usual, and anorexia was complete. During the day he became blind, and the next day about noon he had a convulsion and remained drowsy several hours afterward. The afternoon and evening of the same day he had two convulsions. At 11 P. M. he passed water in a chamber. At midnight he was delirious and required to be held in bed.

I was called to see him on the third day, *October 29*. Temp. 99.4° ; pulse, 96; resp. 34, and puffing. He was apparently unconscious, and passed his water in bed. There was no paralysis. His pupils were equal and responded to light. Both eyes were fixed and turned to the left. The family informed me that about five minutes after I had examined him and left the house, he had had another convulsion. He was seen at 2, 4, and 10 P. M. of the same day. At 4 P. M. he was partially conscious and able to swallow a little brandy and water. He was unable to speak. There was external strabismus of both eyes. During the afternoon and evening his pulse became quite frequent and his extremities cold.

30th, 10 A. M. Temp. 99° in right axilla, and 99.2° in the left; pulse, 150; resp. 32.

Head temperature: Anterior frontal station, right, 97° ; left, 97.1° . Superior frontal station, right, 97° ; left, 97.9° . Posterior frontal sta-

tion, 98° ; left, 98.3° . Rolandic station, right, 98.6° ; left, 98.8° . Parietal station, right, 98.3° ; left, 99° . Occipital station, right, 99° ; left, 99° . No percussion dulness was elicited over the bladder. He seemed to be passing his water freely, but as he voided it involuntarily it was impossible to obtain any for examination. Throughout that day he was semi-conscious, but the next day he became unconscious, and died about midnight. His temperature (axillary) did not rise above 100.5° . His pulse ranged from 150 to 164 per minute. At some of my visits his extremities were cold nearly up to their connection with the body, and death seemed to be momentarily impending; but several hours afterward, at subsequent visits, I found his limbs quite warm. The head temperature was increased more than the axillary, nearly equalling the latter on one or two occasions.

The post-mortem examination was made a few hours after death, before the viscera had lost their heat, by Dr. Strittmatter and myself.

The whole cutaneous surface was jaundiced and greasy, the face and hands having a bronzed hue. Emaciation was moderate, there being still considerable subcutaneous fat. The skin was lax, and could be gathered into folds. The glans penis was hidden by an elongated and constricted foreskin, which could not be retracted. When the latter was slit up, it was found not to be adherent. The subcutaneous and deep lymphatic glands were not enlarged.

Head.—The bones of the head were very thin in some places, and abnormally thick in others, the occipital protuberance being about three-quarters of an inch thick. The occipital bone was so rough and irregular externally in this situation, that an apparent depression existed, but the internal surface of this and the other cranial bones was quite smooth. The dura mater was not more firmly attached than normally. The pia mater was congested, especially over the convexity of the brain, around the fissure of Rolando, where it presented a whitish opaque appearance. Some recently effused lymph was found in this region. The venous sinuses were distended with dark fluid blood, but they were nowhere occluded. The sub-arachnoid space contained about two ounces of serous fluid. After the removal of the pia mater, the entire convexity of the brain appeared to be in a normal condition. The middle and anterior cerebral arteries contained several thrombi of soft, gray, fibrinous material. The vessels were still able to partially perform their function. The left island of Reil and the anterior tip of the left temporo-sphenoidal lobe appeared softer than these structures on the right side. The nerves and other structures on

the surface of the base of the brain, macroscopically, presented nothing abnormal. The ventricles did not contain much more fluid than is usually found in them. The ganglia of the brain appeared healthy. The interior of the cerebrum, crura cerebri, cerebellum, pons, and medulla oblongata, was carefully examined by numerous parallel incisions, but no indication of disease was discovered. The left island of Reil, a portion of the left temporo-sphenoidal lobe, and the pons and medulla oblongata were immediately immersed in Müller's preserving fluid, and subsequently hardened in alcohol for microscopic examination.

Thorax.—The pleural cavities contained only a few ounces of serous fluid. Numerous old pleuritic adhesions were found on both sides, especially on the left. The left pleura was adherent to the upper portion of the pericardium. The anterior upper portions of both lungs were crippled by lobular emphysema, the left to a slight, and the right to a marked degree. Some of the air-sacs were as large as a walnut. At the apex of the left lung was a nodule, about the size of a hickory-nut, whose consistence in its interior was about that of cheese. The bronchial and mediastinal glands were not enlarged. The lower lobes of both lungs were congested posteriorly. The pericardium did not contain more than an ounce of fluid. No adhesions between its layers were found. A small patch of lymph on the left ventricle of the heart, near the apex, was the only evidence of recent pericardial inflammation. The heart, nearly normal in size, was soft, easily torn, and apparently fatty. The right cavities of the heart were slightly dilated, but the left were nearly normal. A mixed fibrous and chicken-fat clot extended from the right auricle into the ventricle, almost completely filling up the latter cavity. A continuation of the same clot was found to reach about four inches into the pulmonary artery. The left ventricle contained some dark fluid blood. A clot, similar in composition to that found in the right cavities of the heart, extended from the left auricle and ventricle into the aorta, a distance of about ten inches, rendering the latter nearly impervious. The valves of the right side presented a healthy appearance, but it is probable that the dilated condition of the right ventricle allowed of regurgitation at the tricuspid orifice. The aortic valves were thickened, and a rim of ossified matter encircled their attached borders. The valves were competent. The orifice was not constricted. The mitral valves were crippled, and rendered incompetent by the ossific change found in them. The size of the orifice had not been much lessened. The incompetency of the valves resulted more from loss of their

pliability due to bony transformation than from their being bound down.

Abdomen.—Considerable fat in the wall of the abdomen was found. No evidence of peritonitis. The peritoneum contained but little fluid. The liver was about normal in size, but studded throughout with numerous cancer-like nodules not larger than hickory-nuts. The growths were chiefly imbedded in the substance of the organ, and did not give it any decided irregular outline when palpated through the abdominal wall. The spleen was about one-half its normal size, tough, and grated under the knife like fibrous tissue. No evidence of disease was found in the pancreas. Each kidney presented the appearance of a large fluctuating cyst about the size of the heart when surrounded by the pericardium. The supra-renal capsules were degenerated, and their contents looked like the fecal matter that is usually found in the bowels at autopsies. The kidneys and their pelves were found to be little more than enormous sacs filled with a watery fluid having but little of the odor of urine. The parenchymatous tissue of the organs was nearly all destroyed. The ureters were dilated to about the size of a man's thumb. The bladder was very small, filled with urine, but it did not reach up to the symphysis pubis. Its mucous surface presented a rigid appearance like the inner surface of the heart. No stricture was found in the urethra. The prostate gland was not greatly enlarged. The bowels were free from obstruction and the stomach from morbid growths. Dr. Henry Leffmann, to whom I sent a specimen of urine taken from the bladder, says: "The specimen of urine was alkaline in reaction and contained a notable amount of albumen but no sugar. The deposit was abundant, of a grayish-white color, and mostly organic. Under the microscope it showed a few phosphate crystals and large numbers of cells, many of them epithelial cells in a state of fatty degeneration. The sediment did not show any evidence of tube-casts or other distinct morphological structure."

Remarks.—The brain complications in this case, as manifested during life, were something more than are found in ordinary cases of uræmia. The partially formed thrombi may be, I think, accounted for by the weak state of the circulation which existed several days preceding death; but the unilateral deafness, the onset of which dated back nearly thirty years, attended by some brain disturbance, the sudden loss of sight and the loss of speech, although consciousness was preserved, point to some distinct nerve tissue alteration. Further, the post-mortem revealed not only congestion of the veins of the brain,

but distinct evidence of subacute pia mater inflammation. Microscopic examinations of the portions of the brain and optic nerve, which I have hardened in alcohol, will throw additional light on the symptoms noted in the clinical history. The elevated surface temperature of the head indicated only a congested condition of the vessels of the cerebral membranes, which probably exists in most cases of uræmia lasting several days.

Two forms of emphysema were present, vesicular and lobular; the former variety was of long duration, as shown by the physical signs recognized years before, but the greater portion of the latter was recently developed owing to the upper portions of the lungs having been violently expanded during the stage of puffing respiration, the lower lobes having become inactive from blood stasis.

In most cases of long standing mitral regurgitation the left ventricle is considerably enlarged; but in the present instance, while the valvular lesion was apparently well marked, neither dilatation nor hypertrophy has kept pace with it. The usual order of sequence following upon valvulitis is, first, disabled valves; second, cardiac enlargement; third, degeneration (if the patient live long enough) of the muscular fibres of the heart. In the heart I exhibit to-night this order in part, at least, was reversed, the degeneration of the cardiac muscle having preceded the valvular affection. The patient had been under my observation a few months before I could detect any murmur, but signs of beginning heart failure were recognized from the first of my attendance upon him. The valvular lesion was the result of the degenerative process going on in various portions of the body, as seen post-mortem, in the heart, aorta, spleen, liver, etc. A left heart already greatly weakened, when any of its valves become incapacitated, because it cannot contract forcibly, is unable to keep up a compensatory hypertrophy, and as its walls are not over distended, dilatation does not take place. In all such cases, as the circulation becomes weaker the strain is thrown upon the right heart, causing it to dilate.

Multiple carcinoma of the liver is said to be almost always secondary to malignant growths in other organs of the abdomen. Authors say its duration rarely exceeds one year. I was unable to find the primary growth, unless the nodule removed from the apex of the left lung prove to be carcinomatous. Was there any connection between the long-continued and deepening jaundice and the hepatic growths? It seems to me that we can only recognize the invasion of the liver by carcinoma, when the physical signs and associate symptoms are present, and that

the period during which the hepatic cells are being encroached upon, before the organ is enlarged, contracted, or rendered nodular, by malignant growths, must escape our notice, except in so far as pain and jaundice may arouse our suspicions. How long the invasion period may be, we have no definite knowledge. I am inclined to think that the duration assigned to carcinoma of the liver is entirely too short. Flint says the average duration is twenty weeks. In the case of the man from whom these specimens were taken, gradually increasing jaundice extended over a period of nearly ten years, and failing health over four years, although no kidney trouble was detected two years before his death. If the jaundice resulted from repeated attacks of hepatic congestion, producing a chronic congestion of the liver, it is probable that this state of affairs made the organ an easy prey to carcinoma. As his father died aged seventy-two years, without any apparent cause, except gradual weakness, it is presumable that he suffered also from internal carcinoma or renal trouble.

The condition of the kidneys represent what is known as pyonephrosis. It undoubtedly was of several years' duration, and probably existed in a mild form two years before his death, although after repeated examinations of his urine, I failed to find evidences of kidney disease. It is probable that the pelves of the kidneys were first dilated by pressure from retained urine, and subsequently, when a chronic pyelonephritis was set up, pus was added. The kidney and bladder troubles were due to the same cause, the former having been secondarily developed. He never suffered from nephritic colic, the ureters were not constricted, no calculi were found in the bladder, and the urethra admitted a good-sized bougie. What, then, was the cause? Authors place phimosis in the list of causative agents, capable of giving rise to pyonephrosis. This deformity of the penis, which was well marked in the man whose history I have given in this paper, was the only probable cause found. I presume, in a case of chronic cystitis, caused by cold, the inflammation might extend up the ureters and involve the kidneys.

The supra-renal capsules had undergone degeneration, and most of their substance had been absorbed. The bronzed condition of the hands and face, associated with the alterations found in the appendages of the kidneys, is the most characteristic symptom of Addison's disease.

Atrophy of the spleen is one of the rarest affections of this organ. It has been very infrequently observed. In this instance, the spleen

was about one-third to one-half its normal size, the decrease being due to atrophy, following hyperplasia of its connective tissue.

In the light of the post-mortem revelations, we need not wonder what killed this man, but rather, how he lived so long.

Report of the Committee on Morbid Growths.—"A section made from one of the nodules of the liver, presented by Dr. Eskridge, examined microscopically, shows it to be a new formation, consisting of epithelial cells placed in alveolar spaces; the tissue forming the alveolar walls being fibrillar connective tissue. The cells in the spaces at the periphery of the groups, and lying next to the alveolar walls, have a columnar shape, and are quite regularly arranged, while those of the centre are flat or squamous epithelial cells. The neoplasms are cylindrical-celled epitheliomata.

"The post-mortem changes undergone by the brain were such as to prevent any satisfactory histological examination being made."

November 9th, 1882.

4. *Microscopical examination of the brain and spinal cord of an epileptic.*

Presented by Dr. CHARLES K. MILLS for Dr. JAMES KINGSBURY.

M. W., aged forty-seven, had had "fits" since she was an infant. During childhood she would have seizures at irregular intervals, sometimes weeks and sometimes months intervening between them. When eight years old, in one of the attacks, she fell into the fire and burned herself severely. After getting married, about the time she became of age, the seizures became more frequent and severe. During pregnancy they would recur as often as once or twice a week.

Some facts in regard to special features of her attacks were obtained from her daughter. They were usually of the grave type of epilepsy, although occasionally between the violent paroxysms she would have spells of *petit mal*, in one of which she fell and broke her leg. Before a grave seizure she would have headache. Just preceding it, she would become giddy, her sight would leave her, and she would usually cry out, "Oh, my head is bad!" Sometimes she would ask for a glass of water, but before she could get it to her mouth, she would fall backward. Her daughter thought that her face was at first pale, and afterward very red or purple. Her eyes would turn upward. She would

usually bite her tongue; and had bitten spoons in two during her paroxysms. She frothed at the mouth, and was convulsed all over; working both hands and feet violently. Occasionally she would pass her urine during the paroxysm.

During the last twelve or fifteen years of her life, the seizures were scarcely ever single, the condition known as *status epilepticus* usually occurring. One fit would follow another for periods of from eight to twenty-four hours. After all the seizures were over she would go into a deep sleep for some hours. On coming out of the sleep she would vomit, and would again have great headache, and would be very weak for a couple of days.

Ten years before her death she was decidedly insane for several weeks, and afterward was "a little queer in the head." During the year preceding her death she suffered greatly with neuralgia, which chiefly affected the right eye. The sight of this eye was very dim, and the ball turned inward. The sight of the other eye was also defective, but less so than that of the left. She was partially deaf in her left ear, and smell and taste were blunted. She had never been paralyzed.

A brother who had died of phthisis had been an epileptic; and a daughter was also the victim of epilepsy, having seizures once in two or three months.

She was admitted into the Women's Nervous Ward of the Philadelphia Hospital, at the beginning of a series of violent convulsions, which lasted nearly two days. She lived ten days after the last paroxysm, but never rallied from a semi-comatose condition.

A post-mortem examination was made four hours after death. External examination showed no scars or evidences of injury. The head was small. The skull was thicker than usual. The brain, after removal, seemed too large for the skull. Little fluid or blood escaped. The weight of the entire brain was thirty-six ounces. A small clot, probably post-mortem, was found in the superior longitudinal sinus. No pachymeningitis was present. The pia mater was everywhere transparent; it showed no signs of old or recent inflammation. The Pacchionian granulations were not perceptible. The brain was of very firm consistence.

The pons and medulla oblongata were smaller than usual. Special appearances were observed on the floor of the fourth ventricle. At the upper part of the right ala cinerea a small extravasation was seen in the substance of the ventricular floor, and on the left of the median

line, in an exactly corresponding spot, the nervous tissue presented a deeply congested appearance. Just below the locus cinereus, on each side, was a small blood-point or spot. About the centre of the floor of the ventricle was a similar appearance. Minute hemorrhagic points were scattered here and there over the floor of the ventricle and the beginning of the crura cerebelli.

Numerous cross-sections were made through the cord. The postero-lateral columns presented yellowish-brown streaks. A small hemorrhagic focus was found in the anterior horn, about one inch below the junction of the cord with the medulla oblongata.

The left kidney was slightly cirrhotic. A small cicatrix was found in the apex of the left lung.

The brain and spinal cord were prepared for microscopical investigation with great care by the following method: A one-per-cent. solution of chromic acid was made. To this was added an equal amount of 95 per cent. alcohol, and the specimens were placed in this solution. A fresh solution of the same kind was used every day for one week, and every other day for a second week. At the end of this time the specimens were placed in strong alcohol. They were kept constantly in the cold. They hardened perfectly.

Blocks of brain tissue were removed from the regions mentioned in the following report, and again put into hardening fluid. The microscopical sections were in those of the convolutions all vertical, and were of sufficient size to show the entire depth of gray cortex and some white matter beyond. Those of the ganglia and tracts were also vertical, and large enough to include the ganglia and capsules. They showed, in other words, in the special sections studied, a part of an optic thalamus, tail of the caudate nucleus, posterior portion of the internal capsule, lenticular nucleus, and external capsule. A longitudinal section of the entire pons and medulla oblongata in its posterior half was made with Dr. Seiler's microtome. With this exception all the sections were cut by hand by Dr. Kingsbury. The spinal cord sections were transverse.

MICROSCOPICAL REPORT.

Anterior end of right frontal lobe.—Everything was normal except an increase of the cells of the neuroglia, in both the gray and the white matter, but particularly the latter, with some obliteration of the perivascular spaces.

Anterior end of left frontal lobe.—Is the same as its fellow, only the obliteration of the perivascular spaces is less marked.

Along fissure of Rolando, right side.—There is a slight increase in the cells of the neuroglia of both the gray and white matter. The periganglionic spaces are not well marked. The perivascular spaces are intact.

Fissure of Rolando, left side.—Just as the right side, but in less degree.

Anterior portion of right cornu Ammonis—gray matter.—Many of the perivascular spaces are narrowed, and some have entirely disappeared. This appearance does not seem to be due to an actual filling up with cells, but probably to dilatation of the bloodvessels. In some places the spaces are intact. The obliterated spaces are apparently limited to certain regions which are mostly peripheral. The periganglionic spaces are barely noticeable, and the ganglionic cells appear to be more granular than normal. The cells of the neuroglia are increased, and appear unusually granular.

White matter.—The obliteration of the perivascular spaces is still more marked, only a few of the larger vessels showing them. The cells of the neuroglia are decidedly increased, and appear granular and cloudy, though the specimen has been most perfectly prepared. This turbidity diminishes as we approach the gray matter, where it is entirely lost. Bloodvessels are dilated.

Anterior portion of left cornu Ammonis—gray matter.—The perivascular spaces are much less obliterated as compared with the right side. The periganglionic spaces are somewhat diminished. The ganglionic cells are more granular than normal. A small saccular aneurism of an arteriole is seen in one of the fissures.

White matter.—The same as the opposite side, only in less degree. Bloodvessels are somewhat dilated.

Posterior portion of right cornu Ammonis—gray matter.—The perivascular spaces are very decidedly obliterated. The periganglionic spaces are not noticeable. The ganglionic cells have a normal appearance. The cells of the neuroglia are increased. In certain places the specimen did not take the staining well, indicating some retrograde change in its elements.

White matter.—The perivascular spaces are less obliterated, but the bloodvessels are much dilated, and many of them are filled with blood. The cells of the neuroglia are increased in number, at some points more than others.

Posterior portion of left cornu Ammonis—gray matter.—The perivascular spaces are less obliterated, some being quite normal. The periganglionic spaces are normal, also the ganglionic cells. The cells of the neuroglia are increased, and somewhat granular. The *white matter* presents the same appearance as the right side, but to a less degree.

Occipital lobe, right side—gray matter.—Increase of the cells of the neuroglia, with slight obliteration of the periganglionic and perivascular spaces, is discernible.

White matter.—There is slight increase of the cells of the neuroglia. Bloodvessels are dilated and somewhat congested. The perivascular spaces are less noticeable than in the gray matter. A remarkable feature is the occurrence in different portions of small capillary abscesses, or rather spots of softening, about the one-fiftieth of an inch in diameter, some more, others less; most of them are located by the side of bloodvessels, which would indicate that they are probably due to capillary hemorrhages.

Occipital lobe, left side.—Affected as the opposite side, except perhaps that the abscesses are not quite so numerous.

Ganglia and tracts, right side.—Same as above, there being numerous abscesses.

Ganglia and tracts, left side.—Same appearances seen, but the white matter is full of small capillary infarcts (each not exceeding one-fiftieth of an inch in diameter), which have not yet undergone softening, as they show distinct masses of blood-corpuscles, which give it a distinctly mottled appearance, as greenish or yellowish spots in the delicate red of the specimen. In some places they have undergone softening. In a limited portion of the gray matter the ganglionic cells are seen to be swollen and pigmented, and appear highly granular. The periganglionic spaces are completely obliterated. A few of the bloodvessels in the same region appear to have undergone amyloid degeneration. In other places the ganglionic cells are swollen and granular.

Medulla oblongata.—There is an increase in the cells of the neuroglia throughout the entire section. The bloodvessels are dilated, and their outer walls are very much infiltrated with cellular elements. The ganglionic cells are swollen and granular, filling up the periganglionic spaces.

Spinal cord, upper cervical region—gray matter.—Normal, except congestion. *White matter* is also congested everywhere. The cells of

the neuroglia are increased in the columns of Goll. On one side of the posterior median fissure is seen a small hemorrhagic infarct, about the one-twenty-fifth of an inch in diameter; also another in one of the posterior roots at its exit.

Middle cervical region—gray matter.—Normal, except two small abscesses in the transverse commissure.

White matter.—Increase of the cells of the neuroglia in the columns of Goll is noted.

Lower cervical region.—Slight increase in the cells of the neuroglia in the columns of Goll is noted.

Middle dorsal region.—Same as the last region.

Lower dorsal region.—Same.

Upper lumbar region.—Normal, except slight increase of the cells of the neuroglia in the columns of Goll, and two miliary abscesses in the transverse commissure of the gray matter.

Middle lumbar region.—Normal.

Lower lumbar region.—Normal.

In all the sections the pia mater was congested.

In all of the specimens, more or less increase of the neuroglia was present. Dilated bloodvessels, obliterated perivascular spaces, diminished periganglionic spaces and granular ganglionic cells were the lesions found everywhere in greater or less magnitude. In the frontal and Rolandic regions these conditions were not marked or extensive.

The investigation of the cornua Ammonis is interesting with reference to the researches of Cazauvieille and Bouchut, Bonneville, Charcot, Delasiauve, Meynert, and Hamilton. Sclerosis, or induration of these parts, similar to that described by the above writers, was certainly found. With reference to these regions, and also all others examined and compared, the pathological changes were more marked on the right than on the left side.

The capillary infarcts, and the minute abscesses or spots of softening (due to capillary hemorrhages) found in the occipital lobes, in the ganglia and tracts, and in the posterior regions of the pons and medulla oblongata, are of great interest.

December 14th, 1882.

5. Tumor of the brain.

Presented by Dr. BRUBAKER for Dr. H. LEAMAN.

John Jones, æt. 53 years and five months; occupation, laborer. When first seen, the patient was lying on his back, with his head drawn backwards into the pillow, and complaining of stiffness and soreness in the posterior portion of the neck. The mouth was widely open and parched, and the breathing deep and heavy. He was in a semi-conscious condition, from which, however, he could be easily aroused, but soon relapsed into his former condition, which was attended by stertorous breathing. Speech and deglutition were both interfered with, but not abolished. The evacuation of the urine was involuntary, but the bowels were constipated. Venereal ideas were excessive, but accompanied with complete impotence. Voluntary movements of the extremities, and also the power of co-ordination were considerably impaired, but not lost. Pulse and temperature were normal. Liquid food was taken with difficulty. This had been his condition during four days previous to my first visit, September 4, 1882. The above symptoms gradually increased. Coma supervened, which ended in death September 17, 1882.

The following history was obtained from the family. Twenty-six years ago, the patient was confined to bed for a period of two years with "nervousness," at the end of which time he passed a calculus about the size of a date-seed, and in the course of a month later passed another of smaller size. His bladder continued to give him more or less trouble up to the time of his death. As the result of an accident nineteen years ago, he had his clavicle broken, but there was no injury to the head. About sixteen years ago, while at work, he was suddenly seized with a severe headache, and became totally blind, which lasted for twenty-four hours. This was relieved by the application of wet cups to the back of the neck. From that time onward he was subject to what they termed "shaking spells;" under these circumstances, when standing, there would be a violent trembling of the knees, and a shaking of the arms. These attacks occurred about once a month, and occasionally three or four times a day. They increased from year to year in frequency and severity, and appeared to be excited by high winds and storms.

In February, 1882, he was seized with paralysis, which began in the left little finger, and then gradually extended to the ring and middle

fingers; the hand became powerless, but he retained the power to move his arm. Then followed a numbness in the outer side of the left thigh, which was attended by an impairment of the power of co-ordination, so that, upon attempting to walk, he was compelled to run, to keep from falling. He frequently fell in this way on the street, and had to be carried home. Last February loss of speech supervened, which lasted for one month; the patient then began to speak in monosyllables, after which speech gradually returned.

A post-mortem examination, made under great difficulty, revealed a general congestion of the entire brain. While removing it, four or five ounces of a serous fluid ran from the cranial cavity. With the exception of the congestion, the brain-substance seemed to be normal throughout. In the right fissure of Sylvius was imbedded a tumor about an inch and a half in diameter, which was almost entirely concealed from view by the convolutions. It rested upon the convolutions of the island of Reil, and completely disorganized them. The inferior extremities of the ascending frontal and ascending parietal convolutions were normal. The upper surface of the temporo-sphenoidal lobe was somewhat disorganized. The tumor appeared to be growing from the pia mater.

No examination was made of any other organ.

Report of the Committee on Morbid Growths.—"A section was made from the tumor of the brain presented by Dr. Brubaker, and examined microscopically; the growth is found to be tuberculous. The histological structure of the growth is seen to consist of fibrous tissue, constituting a reticulum, the meshes of which are filled with lymphoid cells. This arrangement and elements are very distinct at the peripheral zone of the tumor, while the centre and inner zone are in a state of retrograde metamorphosis, presenting a very granular appearance, scarcely stained by the carmine. The bloodvessels are mostly obliterated, their lumen being filled with coagulated blood or granular débris."

December 14th, 1882.

6. *Methodical examination of the brain.*

Read by Dr. CHARLES K. MILLS.

Dr. Mills passed by, with brief allusions, the examination of the scalp, skull, and membranes of the brain, the cerebral spaces and vessels, and the best methods of determining volume, specific gravity,

and cranial capacity. His chief object in the lecture was to discuss and demonstrate the proper procedures to be pursued in methodically examining the surface and interior of the brain, after the removal of its envelopes, for the purpose of discovering and accurately localizing lesions.

In order to make an autopsy of the brain proper in a rapid, methodical, and scientific manner, he considered several requirements necessary.

1. The superficial topography of the brain should be understood. The examiner should be able to pick out, without hesitation, the chief fissures and convolutions. He should also, of course, be perfectly familiar with the numerous subdivisions of the base of the cerebellum.

2. The variations in the size, direction, or arrangement of cerebral fissures and convolutions most likely to occur should be known to him. Age, race, individual type, education, and other causes act to produce variations and anomalies. The student thoroughly familiar with the diagrams and demonstrations of Ecker might find himself at sea in the first examination, if this fact be not remembered.

3. A knowledge of the internal structure of the brain should be possessed by the operator. He should understand not only its ventricles and horns, its commissure and peduncles, its foramina and aqueducts, but also its medullated structure, as exhibited by sections of the cerebral and cerebellar hemispheres made in various directions. Methodical cerebrotomy, practised in the vertico-transverse, vertico-longitudinal, horizontal, and even in other directions, will give certain more or less fixed appearances which the operator should be familiar with.

The surface of the brain should be thoroughly examined for morphological peculiarities, such as deficiency or excess of fissures and gyri, confluence of fissures, simplicity or complexity of folds and furrows; and also, of course, for pathological changes, for atrophy, softening, hemorrhages, changes of color, etc. Dr. Mills described the fissures and convolutions as given by Ecker, and also called attention to some of the most common and striking variations from the usual type of the adult human brain, dwelling particularly on the fissures most likely to be confused; the precentral, central, retro-central, etc. The views and observations of Benedikt on the brains of criminals were referred to and criticized.

The examination of the fissures, convolutions, cranial nerves, and

various regions of the surface of the cerebrum and cerebellum having been completed, the interior of the brain should next claim attention. The examination should be made in such a way as to cause as little disturbance as possible of the relations of parts by the incisions, and to allow, if desired, that the brain be brought together again after the autopsy, and presented as a whole. This could be done so as to submit every cavity, canal, part, and structure to comparatively minute exploration. Some cerebrotomists, as Bitot, without stopping to investigate particularly into the various cavities and subdivisions, at once proceed to divide the brain into blocks, by large incisions made at carefully selected points.

The advantages of each of the three forms of sections—vertical transverse, vertical antero-posterior, and horizontal—were pointed out. The vertical transverse sections are the most generally useful. What are known as the middle transverse, mesolobar, or cortico-central zones are most important to understand. These comprise all the cerebral mass situated between the anterior and posterior extremities of the corpus callosum. Bitot describes and figures seven of these zones. Sections and diagrams were exhibited by Dr. Mills, and the places and methods of making these sections were explained.

The particular convolutions, and the regions of the ganglia, centrum ovale, capsules, etc., traversed by these sections were shown. The ganglio-insular region was particularly described. This region includes a large portion of the island of Reil, of the optic and striate ganglia, of the external and internal capsules, and of the external wall. The importance of having at command a fixed nomenclature for the various planes and regions was dwelt upon. Following the method of Bitot, the surfaces exposed by the sections were divided into a superior, inferior, and middle division; and these were studied. A diagram, representing the relations of the middle portion of the brain to the cranium, was shown and explained.

Dr. Mills said that he preferred to open the brain from below, so as to expose its ventricles, horns, and parts, and examine each as nearly as possible in its integrity. By his method such structures as the corpus callosum, septum lucidum, tænia semicircularis, velum interpositum, choroid plexus, fornix, foramen of Monro, pineal gland and its peduncles, etc., could be, each in turn, investigated for lesions.

After the cavities and horns had been exposed by proper incisions, methodical cerebrotomy, as advised by Charcot and Pitres, or Bitot, could be practised, the cuts being made from within outwards, but not

quite through the brain, so that the whole mass could be drawn together again after the cut surfaces had been examined.

Dr. Mills described three of the best methods of opening the brain: (1) At the base, by taking as guides certain fissures and other anatomical landmarks; (2) From the upper surface, by incisions through each side of the corpus callosum, or along the lower borders of the calloso-marginal fissures; (3) By dividing the brain into two symmetrical halves by a careful median section. This should only be done after the parts in the median line had been previously explored.

Dr. Shakespeare said that he would like the lecturer to state why he prefers to open the brain from the base rather than from above. Dr. Shakespeare thought that by opening from above the floor of the lateral ventricles would be better exposed, which contain the large ganglia of the brain, and a better topographical view would be obtained.

April 26th, 1883.

7. Tumor of the sciatic nerve.

Presented by Dr. G. DE SCHWEINITZ.

The following case occurred in the hospital practice of Prof. John Ashhurst, Jr., and it is by his permission that I exhibit the specimen. The patient from whom this tumor was taken is at present an inmate of the University Hospital, and gives the following history:—

On June 16, 1864, owing to a gunshot wound of the right thigh, he sustained an amputation of that member at the junction of the middle with its lower third. The flaps sloughed, and a few weeks later a re-amputation became necessary. This stump healed, but was somewhat conical in shape and never from the beginning comfortable, being subject to frequent attacks of severe neuralgic pain. Within a year from the date of the amputation a small lump, tender to the touch, was noted, situated posteriorly and a little to the outer side of the stump. The pains now became more severe and more frequent, and were of a "jumping" character, to use the patient's own language. The tumor increased slowly in size until three years ago, when its growth became more rapid and at the same time the painful nature of the affection more pronounced, until lately the suffering was well-nigh unbearable. Finally the growth assumed the size which you see it now presents, and on the 12th of last month Dr. Ashhurst removed it, since which

time the patient has been free from all pain except such as naturally accompanied the healing of the wound.

The growth is an irregularly shaped mass, about as large as a small hen's egg, having an external envelope of adipose tissue loosely held together by connective tissue. On section, the interior is seen to be a somewhat elastic, rather dense-looking growth of whitish color, over which pass a few yellowish fibres, probably strands of the sciatic nerve. Microscopic examination of the true tumor mass shows an entire absence of any nervous elements, a section exhibiting fat cells, fibrous tissue, some spindle-cells, and numerous free nuclei near the enlarged and dilated bloodvessels.

The tumor would, of course, be classed clinically as a neuroma, following amputation, while in truth its pathological nature is that of a fibroma. It is interesting that a growth causing so much pain should be without any demonstrable nervous endowment; and surgically it is further worthy of note, because its removal was attended with immediate and probably permanent relief to the patient, a desired result which is by no means always obtained by the excision of these growths.

June 14th, 1883.

VII. ORGANS OF THE SPECIAL SENSES.

1. *Sarcoma of the capsule of the lachrymal gland.*

Presented by Dr. CARL SEILER for Dr. KEYSER.

The exhibitor explained that he had no history to present except the bare facts that Dr. Keyser had removed the primary tumor May 22, 1881, and a recurrent growth October 21, 1881. Dr. Fenton, however, was present, who doubtless could give some additional points of interest.

Dr. Fenton said that he had nothing special to add, except that when first seen no visual trouble was complained of, but that latterly the eye became displaced by the growth. Until quite recently no pain was complained of. Dr. Fenton did not think that the whole of the growth was removed at the first operation. At present there were signs of a recidive since the operation of October 21, 1881.

December 8th, 1881.

2. *Melanotic sarcoma of the orbit, with metastases to the liver, etc.*

Presented by Dr. SHAKESPEARE.

The patient was an elderly woman, who had been operated upon by Dr. Heyl, at the Episcopal Hospital, some six months before death, the whole contents of the orbit having been thoroughly removed. Recurrence took place, the cavity becoming filled with a black, fungating mass; the left nostril gave vent to a blackish discharge, and the various internal organs became involved, notably the liver. Death took place from exhaustion. Most of the metastases are entirely melanotic, but some in the liver show, at their periphery a distinct, whitish zone. Dr. Shakespeare remarked upon the singular fact that orbital growths were usually melanotic, although they might not spring from the choroid coat of the eye, as in this case, where all pigmental structures had been removed many months ago.

November 23d, 1882.

3. *Several specimens of eyes enucleated to relieve sympathetic irritation in the other eye.*

Presented by Dr. W. S. LITTLE.

Sympathetic irritation and sympathetic ophthalmia are the only two forms of the sympathetic diseases of the eye that afford an opportunity for pathological study, as in these cases only can the enucleated eye be investigated; the exact condition in the eye protected by the enucleation of the diseased one must, of course, remain uninvestigated.

Less and less opportunity is being afforded of studying the condition of an eye enucleated for the presence of sympathetic ophthalmia in the other eye, since merely the symptom of sympathetic irritation in one eye now impels the surgeon to enucleate the primarily diseased eye before true sympathetic ophthalmia asserts itself.

The portion of the eyeball which, when diseased, is liable to produce sympathetic ophthalmia is now so well known that not even sympathetic irritation should be allowed to develop, as an early enucleation will prevent it.

Enucleation of the primarily diseased eye, when true sympathetic ophthalmia is present in the other eye, is now considered questionable, until all inflammation in both eyes has subsided under treatment, since surgical procedures upon the primarily affected eye may afford the best result for visual purposes.

A recent experience will bear me out in the above statements. A patient, refusing the advice of a former medical attendant, and also rejecting my own, after two months suddenly developed sympathetic ophthalmia of the sound eye; enucleation was then too late. At present both eyes are becoming quiet, and I am in doubt which eventually will be the more available eye. A physician, recently under treatment for a seriously injured eye, has refused the advice of two surgeons, and is now doing well; but the danger of a sympathetically irritated eye is constantly before him.

Pathological investigation is then principally confined to examination of eyes enucleated before or after sympathetic irritation has developed in the other eye; when this is done, full protection is usually afforded to the remaining eye.

Investigation of eyes enucleated when sympathetic ophthalmia of the other eye is present may explain the cause of the trouble in the

other eye; but there is usually so much damage done to both, that in the multiplicity of pathological conditions the primary cause is lost.

Eyeballs removed to avert sympathetic irritation, or even incipient ophthalmia, are much better fitted to throw light upon the cause of sympathetic irritation than globes far advanced in disease, since severe inflammatory processes cloud the change from sympathetic irritation to that of sympathetic ophthalmia; and the pathological study is more difficult. My collection only contains one specimen enucleated when sympathetic ophthalmia was actually present; and in this case there was a double acute glaucoma, with a sympathetic iritis.

At this time I only desire to exhibit to the Society some specimens of eyes enucleated to protect the sound eye from sympathetic irritation, or in which it had already developed. In these cases good and permanent results have been attained.

Four of the cases were traumatic; the fifth was inflammatory in character.

All the cases but one of traumatic origin had the sound eye affected, and the examination of the enucleated eye, in this exceptional case, justified the operation. In two cases the sound eye was irritated shortly after the injury was inflicted. In one case, no irritation was produced till forty years after the accident to the enucleated eye. In one case—not traumatic—there was irritability of the sound eye, at times, then subsidence of the symptoms, until enucleation of the diseased eye was required to relieve the great pain in the injured eye and stop the irritation of the other. In the remaining case of traumatic origin, for twenty years there was no irritation of the uninjured eye, excepting that the myopia increased more than is usual; enucleation was demanded on account of the increasing myopia, and for fear of sympathetic irritation or ophthalmia later in life. The ages of the patients were three years, forty years, forty-seven years, fifty years, and seventy years. Three of the patients were males, and two females. In four cases the left eye was involved in the enucleation; in one case, the right eye.

In all these cases the injury or disease involved directly or indirectly the ciliary body. In those cases where the crystalline lens remained *in situ*, or the sclerotic tissue impinged most markedly on the ciliary region, the irritation appeared most rapidly in the other eye. In addition, in those cases where the crystalline lens was dislocated, the irritation appeared late. In one such case with weakening of the

sclerotic tissue, no irritation for forty years appeared in the other eye. In one case with dislocation of the lens and detachment of the retina and choroid, no irritation had appeared in the sound eye at the end of twenty years. When the iris became entangled in the wound as it healed, the irritation appeared very rapidly in the other eye. In one case the lens became cataractous, and in another it was either lost by absorption or was evacuated at the time of the accident. When sympathetic ophthalmia arises in the uninjured eye, it can be then traced to inflammation of the ciliary body, iris, and lens, producing irritation of the ciliary nerves.

It is difficult to determine to what extent the retina and optic nerve participate in sympathetic irritation, since we find that in enucleation for sympathetic irritation the sound eye regains normal vision. In sympathetic ophthalmia, however, the involvement of the nerve and retina is a most important factor, and the sympathetically inflamed eye is not so likely to be restored to its normal condition, as the condition in the enucleated eye and in the one affected is more serious on account of marked inflammatory processes.

With these few remarks, I present the following specimens and the reported cases.

CASE I. White male, æt. 49 years, seen November 22, 1881. When six years of age he stuck a two-tined steel fork in the upper ciliary region of the left eye. He was confined in a dark room for one month; he has refused the advice of surgeons to remove the eye. The left eye—the injured one—has been constantly painful for the past three years. A large staphyloma exists at the seat of injury, so that the eyeball is so large that the lids fail to cover it well, thus giving rise to a superficial keratitis. The cornea is opaque, through which can be seen the crystalline lens cataractous and lying in the anterior chamber adherent to the posterior surface of the cornea.

Tension of eyeball = 1. No perception of light. The right eye has never been troublesome beyond the dislike of looking directly at the light, and then only during the past two or three years. $V. = \frac{15}{xx}$; myopic astigmatism; he has never worn glasses for his presbyopia.

November 25, 1882. Enucleation of the left eye was done. The ocular conjunctiva and capsule of Tenon were adherent to the eyeball.

26th. Wound had healed by first intention.

28th. The vision of the right eye has improved; can bear light well, and spasmodic action of the orbicular muscle has ceased.

December 6. Artificial eye given.

November 24, 1882. I examined the enucleated eye, it having been in alcohol. The antero-posterior diameter is one and a quarter inch, equatorial one inch. On section, I found the retinal chamber full of what appeared to be pus; there was no division between the chambers of the eye; the ciliary body was disorganized, the retina adherent to the choroid; the crystalline lens was in the anterior chamber adherent to the cornea; the sclerotic was thinned at the seat of the wound. The sympathetic irritation did not appear in this case until some forty years had elapsed, and not until severe cyclitis was excited.

CASE II: Colored male, *æt.* 40 years; seen October 10, 1882. Had the left eye cut open during a fist encounter in July, 1882. The cut was V-shaped, and was in the middle of the cornea extending upward to the ciliary region; the crystalline lens was lost at the time. When seen the eyeball was tender and shrunken.

Soon after the eye was hurt, the sound eye—the right—which had been injured when a child by having the cornea cut with the edge of a blade of corn, and kerato-iritis having resulted, took on sympathetic irritation.

The symptoms were very marked when I saw him. Immediate enucleation was advised and done. A good result followed. An iridectomy, to be done later, will give more effective vision to the right eye on account of the central corneal opacity.

Examining the specimen, you will see that the ciliary body is pressed upon by the sclera in its whole circumference; the force of the blow and the subsequent contraction flattening the sclera upon itself in the anterior portion of the eye, so that the specimen looks very much like a cooked mushroom. The iris is entangled in the corneal tissue at the seat of the cut. The retina was detached. The condition of this eye explains the cause of irritation, and the already diseased state of the right eye soon caused sympathetic trouble.

CASE III. Male child, *æt.* 3 years. Seen June 1, 1882, at the Jefferson Medical College Hospital. The left eye was cut in the outer sclero-corneal margin—the wound extending slightly into the cornea—by a piece of glass which penetrated the eye.

The patient was seen two weeks after the accident, when severe kerato-iritis and traumatic cataract existed. The right eye was only slightly irritated then, but on June 14, 1882, I was compelled to enu-

cleate, the eye injured not getting quiet on treatment and threatening the loss of the other eye.

The specimen, opened at the time, showed existing cyclitis, hemorrhage into the vitreous, and the lens cataractous.

CASE IV. Colored female, æt. 70 years. She was seen in June, 1882. I found the woman in great pain from a glaucomatous condition of the left eye, secondary to what I thought was a sarcoma of the choroid or ciliary body. The eyeball was much enlarged, and a protuberance existed at the equator above—this may occur in glaucomatous eyes, and can be seen when rotating the eye downwards. It extended about one-half inch above the normal curve in that position. She stated she had been troubled with the eye for several years, and two years ago was in an eye hospital, and fearing to take ether, she had declined the operation without an anæsthetic. At that time I presume the diagnosis was in doubt. The right eye was irritated. June 22, under ether, I removed the left eye. I had some difficulty in getting it out on account of its shape. Some reaction followed the operation as the contents of the orbit had been irritated by the size of the eyeball and the manipulations for its enucleation. The remaining eye was much relieved by the operation, and there has been no pain in it since. I saw her lately, and found her greatly troubled with asthma and general dropsy. She says that she is losing the sight in the remaining eye. She has evidently senile cataract. The enucleated eye was recently examined. I found that in alcohol it had shrunken much, and on section I discovered not what I had expected, viz., a sarcoma of the choroid, but an inflammatory mass involving the ciliary region. From its position I presume it was due to inflammation of the vitreous body: the ciliary region was impinged upon. The retina was detached posteriorly. The specimen is quite unique, and I am not able to explain the exact condition present, or why the protuberance should have been where it was prior to removal, as the globe now presents such an uniform appearance around the ciliary region. The eye was entirely blind and in a glaucomatous state when operated upon.

An ophthalmic view of such a case, early seen, might be confounded with glioma of the retina.

CASE V. White female, æt. 47 years. Seen April, 1882. The right eye received a blow some twenty years ago, dislocating the crystalline lens into the anterior chamber. Vision was lost, and the lens

became cataractous. The eye has been irritable at times. At the point where the lens touched the posterior surface of the cornea there was an opacity and a superficial keratitis had been excited when I saw her. The left eye was highly myopic and astigmatic. $V. = \frac{1}{\infty}$. I advised enucleation, after correcting the vision in the remaining eye with glasses, for fear of irritation, which was not present, or if so, to a very slight degree.

June 5, 1882. Having watched the case for a few months I removed the right eye. L. E. V. = with glasses $\frac{15}{\infty}$; a large posterior staphyloma was present reaching to the region of the fovea centralis, which rendered better vision impossible.

I examined the eye recently, after being in Müller's fluid, and it presents slight detachment of the retina, some inflammatory changes at the region of the ciliary body, and the lens is in the anterior chamber.

The case is interesting in connection with Case I.

December 14th, 1882.

VIII. TUMORS NOT OTHERWISE CLASSIFIED.

1. *Case of congenital cyst of the neck.*

Related by Dr. J. H. MUSSER.

The tumor was situated on the right side of the neck ; it was as large as the child's head ; the base extended from the median line of the neck anteriorly to within an inch of the posterior median line, and from the edge of the lower jaw to the clavicle. The sac was distended, the wall quite thin ; on the surface there was great capillary injection, but there were no evidences of a previous nævus. The fluid drawn off was straw-colored, clear, and contained albumen. In three days it re-filled. Professor Agnew saw the child with me, and advised the introduction of a seton. The seton set up inflammation of the wall of the sac ; it became thick, while the discharge changed from serous to sero-purulent, and then a thick, grumous, yellowish-green, very offensive fluid. The sac would diminish in size, to re-fill again, especially if the counter-outlets, which spontaneously opened, happened to close. In two and a half months the discharge ceased entirely, and at present there are three or four puckered and depressed cicatrices in the middle of the neck, with considerable redundancy of the skin around them.

I should have stated that the child had hereditary syphilis, and that I had it on mercury for some time. *January 12th, 1882.*

2. *A tumor composed of miliary tubercles of the subcutaneous adipose tissue connected with one of the anterior cutaneous branches of the lumbar nerves.*

Presented by Dr. NANCREDE.

The patient from whom this truly unique tumor was removed was a young girl 18 years of age, who for four years past had had occasional cough, with at times some bloody expectoration, but who was able

to attend to her occupation of housework. Her family history was not characteristic in any way. About one year since she thought that she "strained herself," since then she has been subject to severe attacks of abdominal pain, which extend to various portions of her body. She was admitted to the female medical wards of the Episcopal Hospital last fall, where dulness on percussion and harsh respiration at the apex of one of the lungs were detected. During December, 1882, the pains increased and the right thigh became flexed upon the abdomen. A small, exquisitely sensitive, nodulated tumor was now detected just to the outer side of the right rectus abdominis. Dr. Morris J. Lewis, by whose kindness I am enabled to present this specimen, then asked me to see the case with him. Under ether I found a nodulated mass, beneath, but attached to the skin, and freely movable upon the deeper parts. I then thought that the growth was one of the ordinary so-called neuromata, *i. e.*, usually fibrous growths in connection with some nerve, and that the abdominal pains were reflex, as was also the flexion of the thigh. February 20, 1883, I accordingly removed the growth, which to my surprise was markedly infiltrated, and only at one spot in any sense encapsulated, where it evidently had developed around a small cutaneous nerve and artery. The wound did badly, and has left an indolent ulcer, but *all the reflex pains and flexion of the thigh have disappeared*, while the lung is breaking down; yet the patient is gaining flesh, and looks and expresses herself as much better, and thoroughly satisfied with the results of the operation. I have termed this growth "unique" because I believe that none such have been reported, *i. e.*, subcutaneous masses of tubercle large enough to require the surgeon's knife, and liable to be mistaken for other neoplasms. The present growth was about an inch in its various diameters, as far as could be estimated. Microscopically, sections show fibrous and adenoid tissue, with giant cells, according to the kind report of my friend, Prof. Simes, whose observations have been confirmed by Dr. Formad and other pathologists, as well as by myself.

Dr. W. G. Davis said that he had seen a somewhat similar case in the clinic of Prof. König, of Gottingen. A young man had a subcutaneous tumor just above and to the outer side of the patella. It was about one inch and a quarter in diameter, and perfectly circumscribed. It and a portion of the joint-capsule, including the part to which it was attached, were excised antiseptically. On the synovial membrane were found a number of what appeared to be miliary tubercles. There were no other evidences of tubercular disease, and he

recovered with a good movable joint. Prof. König regarded the case as one of true localized tuberculosis. He examined the excised portion microscopically. The tumor was hard, but had undergone cheesy degeneration.

Dr. Nancrede thought that this interesting case related by Dr. Davis still left his own unique, as in Prof. König's case the tumor evidently had its origin in the synovial membrane, which was so closely related to the other serous membranes, which, as is well known, are so very prone to miliary tuberculosis.

March 8th, 1882.

3. *Epithelioma of the leg.*

Presented by Dr. H. F. FORMAD for Dr. W. G. PORTER.

The following history was kindly furnished by Dr. Ch. W. Kollack:—

Rose D., born in Ireland, age 43 years. She twisted her ankle on a curbstone, but felt very little pain at the time. At the expiration of two or three weeks the leg began to swell, and considerable pain was experienced. It was several years later before there was any sore, and after this occurred a piece of bone came out; the sore healed, but was not "solid," as she expresses it. Later on, the sore again opened, and had the appearance of "proud flesh." She entered the Episcopal Hospital, and was operated upon by Dr. Ashhurst, who removed a portion of the tibia. The leg never healed after the operation,—that is, entirely. She entered the Philadelphia Hospital in February, 1881, and from that time the leg grew steadily worse until the 4th of January, 1882, when it was amputated by Dr. W. G. Porter just below the tubercle of the tibia.

It is now about fourteen years since the ankle was first injured. Before the accident she was perfectly healthy, and her parents were healthy according to her statement. The patient is now doing well, but the stump is not healing.

April 12th, 1882.

4. *Spindle-celled sarcoma of the thigh.*

Presented by Dr. J. HENRY C. SIMES.

The patient from whom this specimen was removed presented himself for admission to the Episcopal Hospital on August 30, 1882. He

was 60 years old, an Irishman, and gave the following history: Two years ago he first noticed, at the lower and outer part of the thigh, a small swelling, which grew rapidly to the size of a hen's egg, when it was removed by a surgeon. Shortly after the wound caused by the removal of the growth had healed, a second tumor was noticed in the cicatrix, having the same characters as the previously removed growth. This was also removed by an operation. And, again, in a still shorter interval, a third similar growth was developed in the same locality, which was treated as the former tumors.

At the time of his admission into the hospital there was found, upon examination, at the lower and outer part of the thigh a linear cicatrix about two inches long. Beneath and adherent to this cicatrix, as well as to the surrounding integument, was seen a tumor as large as a walnut; it was movable upon the deeper tissues, painless, dense, and irregularly nodular. There was no glandular enlargement observable. No other growths were found when the entire surface of the body was examined. The tumor was readily removed; the adhesions were only to the overlying skin which was included in the removal.

Microscopic examination showed the neoplasm to consist entirely of large spindle-shaped cells, which contained large oval-shaped nuclei.

September 14th, 1882.

5. *Lymphomatous tumor of the mediastinum.*

Presented by Dr. W. S. LITTLE for Dr. G. C. SMITH,
of Rondout, N. Y.

The history of the patient from whom these specimens were removed, is, in brief, as follows: A young man, æt. 24 years, had been but a few days under Dr. Smith's care, having come from Boston, where his physician had pronounced him phthisical, and had recommended a sea-voyage. During the past few months small, nodular masses had developed in the muscular tissues of the right chest-wall, near the median line in front, and also posteriorly. The axillary and supraclavicular glands were involved. Shortly after Dr. Smith first saw him he developed marked dyspnœa, and died suddenly, without any evidences of marked lung disease, except, perhaps, some symptoms of pleuritis. There was apparently mitral disease. Anasarca, especially of the lower extremities, gradually developed.

Sectio cadaveris.—The skin was hard and friable; nodular masses were found disseminated through the muscular tissue of the chest-walls, which had undergone some species of degeneration; the costal cartilages presented evidences of a degeneration similar to that seen in the muscles. On removing the sternum, the subjacent tissues were markedly pigmented, and the anterior mediastinum completely obliterated by a mass of the size and shape of the half of a large lemon, which pressed against the heart. The large bronchi were involved in the growth; otherwise, the lungs seemed healthy, and evidences of slight pleurisy were found. The pneumogastric nerves were both involved, chiefly the left, and on further dissection the disease was found to occupy all of the lower part of the posterior mediastinum, involving the contiguous osseous tissues. The nerve involvement explains the sudden death. The diaphragm was also involved in the lower portion of the growth. The four portions of the growth shown to the Society are: 1st, one of the subcutaneous nodules; 2d, a small portion of the left lung, near its root; 3d, a portion of the anterior mediastinal growth, with part of the trachea, bronchi, and aorta; 4th, one-quarter of the tumor, which, involving the diaphragm, projected from the left thoracic wall into the chest-cavity.

Dr. Formad inquired whether there were any other evidences of carcinoma in the remainder of the body.

Dr. Little replied that none were detected.

Dr. Formad then said that he was unaware of any specimen of primary carcinoma of the mediastinum on record, and moved the reference of the specimen to the Committee on Morbid Growths, as it was probably a sarcoma.

Report of the Committee on Morbid Growths.—"The mediastinal growths are found, on microscopical examination, to consist of a mass of hypertrophic lymphatic glands, much pigmented. There are also seen adipose and fibrous-tissues in a state of active proliferation."

October 26th, 1882.

6. *Myxomatous tumor of the posterior cervical region.*

Presented by Dr. NANCREDE for Dr. W. G. MACCONNELL.

The tumor was removed by Dr. J. H. Brinton, at the Jefferson College Hospital Clinic, some ten days since. The patient was a little boy, aged four years, whose parents had first noticed the growth about

two years ago. Latterly it has grown with considerable rapidity. It was of firm consistence, lobulated and movable beneath the skin, giving the impression that it was a fibrous tumor. After removal, in addition to the above-mentioned characteristics, it was found surrounded by a capsule, and on section looked somewhat suggestive of myxoma, still it was thought by some to be merely a fatty tumor, containing more fibrous tissue than usual.

Microscopic examination by Dr. MacConnell.—Upon examining a frozen section stained with iodine, meshes of capillaries are discerned, in the walls of which the endothelial cells composing the vessels can be distinctly seen. The aforesaid meshes contain the mucoid structure traversed by large, pale, fusiform cells, the processes of which anastomose with each other. In addition many leucocytes are seen, and interspersing the growth, in every direction, numerous yellow elastic fibres are readily distinguished.

When presenting this specimen Dr. Nancrede commented on the rarity of such growths, and referred to a hæmatoid myxoma of the breast, presented to this Society some years ago by Dr. Barton.

Dr. S. W. Gross said he had himself presented several gelatinous polypi of the nose, a number of years ago, which were most characteristic examples of myxomatous tissue. He could also recall a specimen of subcutaneous myxoma of the forearm, as well as the hæmatoid myxoma of the breast referred to by Dr. Nancrede. He was disposed to consider it the rarest of all neoplasms of the breast; indeed, he had never personally met with one, and when preparing his work on tumors of the breast, he had written to numerous surgeons throughout the country, who all replied that they had never met with one affecting the breast.

Dr. Formad remarked that he had exhibited a myxomatous fibroma of the labium some years since, and said that the peculiar milky appearance assumed by the fluid when such growths were thrown into alcohol was a good diagnostic point.

Dr. Shakespeare said that his personal experience as to the rarity of myxomata coincided with that of Dr. Gross. This specimen is one of the rarest forms, as most of the fibrillæ consist of yellow elastic tissue. The rarity of myxomatous tumors seems to him to have much bearing on the views of Cohnheim and others as to the etiology of tumors. These observers insist that all tumors spring from the remains of foetal tissues not made use of in tissue construction, which remain dormant in their embryonal condition until subjected to some irritation, when they de-

velop into the various neoplasms. Now, tissue practically identical with that found in myxomata pervades the fetus. How, then, is it that portions of this do not remain, to give rise to myxomata? On the contrary, myxomata are among the rarest of the neoplasms.

November 9th, 1882.

7. *Congenital fatty tumor beneath the occipito-frontal muscle.*

Presented by Dr. NANCREDE.

The tumor was simply presented on account of the rarity of such growths of *fœtal origin*. The patient was a child, aged 16 months. The growth was noticed three weeks after birth, and in consequence must have been of fœtal origin, as it then was of the size of a bean. It presented none of the symptoms of a fatty tumor, except a faint lobulation, the skin moving freely over it, and showing none of the dimpling so common in lipomata. Its site was peculiar for a dermoid cyst, viz., over the right occipito-parietal region, yet its resemblance was so close, that by exclusion it was considered to be a congenital cyst. Upon removal it was seen to be beneath the aponeurosis of the occipito-frontal muscle. Microscopic sections, kindly made with the freezing microtome by Dr. W. G. MacConnell, showed that the growth was a pure lipoma.

May 24th, 1883.

IX. SPECIMENS FROM THE LOWER ANIMALS.

1. *Myxomatous tumor of the mesentery of a hen.*

Presented by Dr. F. P. HENRY.

Dr. F. P. Henry presented a hen in which the mesentery was the seat of a morbid growth closely resembling myxoma in its gross appearance. The history of the case was entirely post-mortem. The morbid growth attracted attention at one of the markets, and the entire carcass of the fowl, with the tumor *in situ*, was carried by a gentleman who obtained possession of it to the Philadelphia Dispensary. One of the physicians connected with that institution referred him to Dr. Henry as one likely to be interested in the subject; and to this gentleman, whose name could not be ascertained, the Society is indirectly indebted for the opportunity of beholding what is, without doubt, an exceedingly curious and interesting specimen.

As the specimen was only obtained by Dr. Henry a few hours before its exhibition, he had had no opportunity to give it a thorough microscopical examination. A partial examination, however, demonstrated that the stroma was composed of dense connective tissue, and that the fluid occupying the cellular interspaces was very poor in solid matters, a few oil-globules and blood-cells being the most conspicuous, and these probably came from the stroma.

The growth appears large enough to have produced some deformity of the fowl during life; but on this point nothing could be ascertained. The liver was healthy, and the tumor was not ovarian. It corresponded precisely to the mesentery, the intestine occupying its convex borders. There was no intestinal obstruction. On first sight the appearance presented by the mass closely resembled that of an extreme emphysematous infiltration of loose connective tissue; but the sense of touch immediately corrected this idea. The growth was firm, non-crepitant, and elastic.

At Dr. Henry's request, the specimen was referred to the Committee on Morbid Growths.

Report of the Committee on Morbid Growths.—"A microscopic examination of the tumor of the mesentery of a hen, presented by Dr. F. P. Henry, showed that it presented the structure characteristic of myxoma."
September 23d, 1881.

2. *Carcinoma of the mammary gland of a dog, with metastases to the liver and lymphatic glands.*

Presented by Dr. H. F. FORMAD.

An old female rat-dog, about two years ago, showed a circumscribed induration in one of the mammary glands, of the size of a walnut. This subsequently proved to be a permanent tumor, increasing very slowly in size, and causing the animal to suffer, at times, intense pain. One year ago, when I saw it first, the tumor was of the size of a hen's egg, and painful on pressure. There was also involvement of the axillary glands, which were large and nodulated. I did not see the dog again until a year later, when it died and was kindly sent to me by Dr. Keys.

Autopsy.—The animal was greatly emaciated. The mammary tumor had reached the size of a large orange. It was encapsulated, round and flat in shape, hard, and the skin was movable over the tumor. All over the abdomen and chest there were seen about twenty secondary tumor-nodes, varying in size from a pea to a hazel-nut. The axillary glands were nodular and greatly enlarged through infiltration by the new growth. Several secondary nodes were found in the liver. There was also a remarkable enlargement of the thyroid gland, which on section proved to be an aneurismal goitre. Microscopic examination of the mammary tumor and of the secondary deposits showed scirrhous carcinoma.

Dr. S. W. Gross remarked that if this growth was truly carcinomatous, it certainly did not resemble the same neoplasm in man, as it was clearly circumscribed by a distinct capsule.

Dr. Tyson was a little doubtful whether it could be really carcinoma, even with the microscopic appearances described, since macroscopically the growth differed entirely from human carcinoma, as it did not infiltrate the surrounding tissues.
May 25th, 1882.

X. MISCELLANEOUS.

1. *Address of the Retiring President, Dr. S. W. Gross.*

Delivered November 9th, 1882.

Having, during the first two years of my term of office, complied with the regulation requiring the President to sum up the work of the Society during the previous year at the annual meeting in October, I shall on the present occasion detain you only a few minutes in summing up the work of the past year, and in contrasting the condition of the Society with that in which I found it upon assuming my duties as your President three years ago.

The meetings were held regularly, and were fairly attended, the average having been 15, as against $13\frac{1}{2}$ for 1881; and the specimens presented having numbered 57, as against 47, during the same period. At one of the meetings in October, the paper of Dr. Formad on the Etiology of Tumors, read during the previous year, was discussed, and at the conversational meetings highly instructive and valuable papers were read by Dr. Bartholow and Dr. Cohen. During the year a handsome volume of Transactions has appeared, making the second published during my term of office.

During the year 9 members resigned, 11 were dropped for non-payment of dues, 5 died, and 41 were elected, leaving a resident membership of 164, or 1 more than when I entered upon the discharge of my duties, despite the fact that during my three years' service, we lost 48 members, 29 by resignation, 8 by death, and 11 being dropped from our rolls. The majority of those who resigned or were dropped had not attended a meeting for several years, so that the working force of the Society is larger than it has ever been in the twenty-five years which have elapsed since it was founded. During the year we have had to regret the loss by death of Drs. R. M. Bertolet, T. H. Cathcart, Wm. Darrach, F. C. Hand, and W. F. Jenks.

The income of the Society during the past year was \$825.07, a larger sum than was ever before received, and the expenditures were \$561.80, leaving a balance on hand of \$401.79, against which \$565.33

are chargeable, to meet which back dues to the amount of \$135 can be collected, and \$750 should be received from dues during the present year. Had it not been that we were paying off our debts, we could have started the new year with a clear record. As it is, the Society is to be congratulated upon its sound financial condition, for which we are indebted, mainly, to the untiring efforts of our Treasurer.

From these data it will be perceived that the Society enters upon the second quarter of a century of its existence in the most flourishing condition. To maintain it at this standard, and to increase its further usefulness, should be regarded as the work particularly of the younger members, as the older ones are actively engaged in the arduous duties of a professional life, and cannot be expected to be regular attendants upon the meetings, especially when we take into consideration the large number of societies which exist in our midst, and which make certain drafts upon one's time. In fostering the interests of our body, I would respectfully impress upon the members the importance of casting aside all personal feelings in the selection of their officers, since we were in danger at one time of being wrecked by just such an unworthy consideration. Above all, they should not be actuated by feelings of rivalry between our two great medical schools, but distribute the offices as evenly as may be between the graduates of the two schools, and thereby cultivate closer ties of friendship. In conclusion, I have to thank you for the courteous manner in which you have dealt with my shortcomings as your presiding officer, and to say that I relinquish my position with the kindest feeling toward all of you. As the oldest elected resident member of the Society, having joined it a year after its organization, or twenty-four years ago, I can assure you that I shall always take the liveliest interest in its welfare, and attend as many meetings as my professional duties will admit of.

2. *Address of the Retiring President, Dr. James Tyson.*

Delivered October 11th, 1883.

It is perhaps fortunate for your President that the scope and objects of his annual address are prescribed by the constitution, which requires that "he shall sum up the work of the Society for the past year, and make such suggestions with regard to its labors, and the mode of conducting them, as may seem to him desirable."

There has probably been no year in the history of the Society in which the number of specimens was as large, there having been exhibited at 18 meetings 80 specimens, or an average of $4\frac{1}{2}$ for each meeting; while, during the previous year, but 49 specimens were shown in the same number of meetings, or an average of $2\frac{3}{4}$.

Some of the specimens were of more than usual rarity, and among these should be especially mentioned:—

Cirrhosis of the Liver with Hypertrophy, by Dr. Bruen.

Tongue and Larynx, from a case of Elephantiasis Græcorum, by Dr. Beecher.

Atheroma of the Pulmonary Artery, with Opening in the Septum of the Ventricles, by Dr. Bruen.

Melanotic Sarcoma of the Orbit, with Metastatic Deposits, by Dr. Shakespeare.

Secondary Sarcoma of the Heart and Lungs, by Dr. Willard.

Miliary Tuberculosis of the Omentum, by Dr. Formad.

Diaphragmatic Hernia, by the President.

Collections of Miliary Tubercles from the Subcutaneous Tissue near the Umbilicus, by Dr. Nancrede.

Metastatic Pneumonia following Pistol-shot Wound of the Temporal Bone, by Dr. Fisher.

Tympanic Caries producing a subdural abscess which opened into the lateral sinus, by Dr. Davis.

Carcinoma of the Rectum, associated with Sarcoma of the Bladder, by Dr. Musser.

Hæmatoma of the Mediastinum, by Dr. Eskridge.

Congenital Lipoma removed from the Head of a Child, by Dr. Nancrede.

Spindle-celled Sarcoma of the Small Intestine, by Dr. Wm. A. Edwards.

Sub-epithelial Clot in the Stomach, by Dr. Barton.

Secondary Sarcoma of the Heart and Lungs, by Dr. Willard.

Fibrous Myxoma from the Posterior Cervical Region, by Dr. Nancrede for Dr. MacConnell.

The descriptions which have accompanied the specimens exhibited have been for the most part appropriate, but in a few instances members have allowed themselves to digress rather more than is demanded by the objects of the Society, or than can be permitted, if the number of specimens to be exhibited continues as large as it has recently been.

These digressions have been in two directions: one in that of a too lengthy clinical history of the case whence the specimen is derived; and the second is the reading of what might be called essays or papers on the subject illustrated by the specimen, in which authorities are quoted at considerable length, and a general discussion of the subject entered into.

While it is desirable and necessary that a certain part of the clinical history of each case should be given, generally as much as can be recalled by memory, if the case has been under the care of the member presenting the specimen, or from brief notes, if the specimen be exhibited by another, the daily notes of a case in no way help us to understand the specimen, while it becomes irksome to the audience to listen to them.

The second form of digression, that of the essay or paper, would be quite appropriate, if the number of specimens exhibited was smaller, or our time more unlimited. But the former having become as large as at present, with a reasonable expectation of an increase which should be in every way encouraged, the description should include only such collateral information as bears directly on the matter in hand. On the other hand, the specimens themselves are sometimes not described with sufficient minuteness, and our own discussions are not as full as they ought to be. At the same time, it must be evident to the most careless observer, that a much larger number of our members do show evidence of having a knowledge of pathology than was the case a few years ago. Many of the facts which have heretofore been embodied in the essays or papers referred to, might be brought out in the discussions, which would thereby be rendered more interesting, as well as fuller.

Another reason for a suitable brevity in the paper accompanying the specimen is, that the class of medical journals in which we prefer to see our "Proceedings" published, is always pressed for space, and their editors naturally do not care to have it occupied by lengthy clinical histories which have nothing unusual, or especially useful to their readers.

The average attendance at each meeting was 20.

Another gratifying feature in the Society's history, during the past year, is the large accession to membership, and the average attendance. From September, 1882, to June 30, 1883, 28 members were added to our roll; 1 died, and 2 resigned, making the total number

of members at the beginning of the session 1883-4, 158, or an actual gain during the session of 25.

The death of Dr. John Forsyth Meigs has removed from our rolls one of the most distinguished members, and one of the original numbers who founded the Society in 1857.

The finances of the Society are also in a very healthful state. At the beginning of September, 1882, the Society was in debt \$163.54. On September 1 of this year there were in the treasury \$184.90, with charges against it of \$150, leaving a balance of \$34.90. There were also due the Society, of annual dues, \$256, much of which is collectable.

But one conversational meeting was held during the past year. At that our fellow-member, Dr. Charles K. Mills, gave us an admirable lecture on a subject on which he is peculiarly qualified. It is that of post-mortem examination of the brain, than which none is more important, and regarding which the profession is sadly deficient. Almost no one knows how to make skilfully an autopsy of the brain, and yet there is no part of the body, a correct examination of which is as important. It appears to me that for a time Dr. Mills might repeat his lecture annually, with great profit to the members of the Society. This meeting was followed by a reception and inexpensive collation, which were deemed a success. The cost was defrayed from the treasury of the Society. The conversational meeting which should have been held on the fourth Thursday in September was omitted because the Business Committee found it impossible to secure a lecturer for that date. They have, however, provided for such a meeting to be held on the evening of Thursday, October 25, when Dr. Carl Seiler will deliver a lecture upon Posterior Nasal Hypertrophies.

The time set down for this semi-annual conversational meeting, as provided for in our constitution, is an unfortunate one. As one meeting occurs the fourth Thursday in April, and another on the fourth Thursday in September, immediately after the long summer recess, it is extremely difficult to secure a lecturer during this interval, and I would suggest that the constitution be amended, to provide but a single conversational meeting in the year, which might be held in January or in February. This could be more easily filled, and followed by the usual reception, and altogether the occasion might be made one of greater éclat than according to our present arrangement, although it must be said that the conversational meetings in the past have proved great successes, contributed greatly to the prosperity, usefulness, and

reputation of the Society, which is universally regarded as the most important of its kind in the United States.

And this brings me naturally to the consideration of the volume of Transactions or Proceedings, as it strictly is. There is no doubt that the reputation of our Society abroad is due to the publication of this volume, which is a most valuable store-house of pathological knowledge, second only to that contained in the Transactions of the Pathological Society of London. It is nothing against such a volume that there is not a large sale for it. It is not a book to be read, but a work of reference, and therefore useful mainly to the student, and to be found in his library, and in the general medical library. It appears to me that the first object of the Society, after the exhibition of specimens, should be to publish an annual volume. It is the most enduring monument that the Society can erect to itself, and all our ends should be shaped towards it. I hope, therefore, that the Society will see its way clear to immediately preparing the accumulated material of two years for publication.

The Treasurer furnishes me a statement, according to which the prospective income of the Society for the ensuing session is \$1072. The expenses \$500, leaving a probable balance of \$572 towards the expenses of the volume. The previous volume, IX., cost \$500, and has been paid for chiefly out of the income of the past session, although the Society began the year \$163.54 in debt, while we began the present year with a balance of \$34.90 in the treasury.

The prize of \$25 for the best exhibit with report of pathological specimens was awarded to Dr. Guy Hinsdale, at the time a resident physician at the Episcopal Hospital.

This sum has been profitably expended, and I think we might well afford to repeat it, although it may be desirable to condition its award upon there being more than one competitor.

In conclusion, I would again remind the members that the Pathological Society of Philadelphia has earned for itself a reputation second to none in this country, and that it becomes us to make every effort to keep the advanced position we have earned. Although the number of specimens exhibited has been large, it may still be larger without overcrowding us, and I have no doubt many specimens of rare value have escaped the attention of the Society, which might have been secured to us, if we had been willing to take a little extra trouble, and had but the courage to ask for autopsies in cases which

have died under our care. There are still too few autopsies made in private practice, and many more could be secured for the asking.

As far as my own experience goes, I have never failed to be more than repaid for the personal annoyance it has cost me to secure an autopsy.

3. *Some comments on the etiology of tumors.*¹

Read by Dr. H. F. FORMAD.

In the paper which is the subject of discussion this evening, I endeavored to prove the proposition, viz., that all *primary* tumors, save the purely congenital neoplasms, are direct products of the inflammatory process.

A certain class of tumors are admitted by several pathologists to be due to inflammation, while to this cause I ascribe nearly all tumors. Again, those pathologists regard inflammation only as an exciting cause, provided there is a predisposition to tumor-formation. I am inclined to regard the inflammatory process as the factor which creates this predisposition, and hence consider inflammation as a direct predisposing cause for all true tumors. This is the difference between the view held by the real authorities of the inflammatory theory—S. D. Gross, Virchow, and Samuel—and the view which I advocate.

The idea of an inflammatory origin of tumors begins of late to gain more and more ground among working pathologists. Several of the true tumors are nearly generally admitted to be due to inflammatory causes, and although no one expresses himself decidedly upon the subject, I do believe that all will ultimately return to the view which the fathers of pathology originally held.

My studies on the etiology of tumors are by no means completed; still, I bring the work forward in its present state in order to get the full benefit of criticism. I want advice and co-operation; I desire to learn whether the new facts which I have obtained by microscopic and other studies admit of an interpretation different from that which I have given them. At any rate, I consider my work only an attempt at the solution of the question of the etiology of tumors, a

¹ The paper has been published in full in pamphlet form by order of the Society. An abstract of it, entitled "The Inflammatory Origin of Tumors," appeared first in Seguin's "Archives of Medicine," October, 1881.

question which is so much neglected, and which imperatively demands active work, and not hypotheses.

Before I enter into a review of my arguments, I desire here to call attention to and to define more closely the purely congenital anomalies called "tumors," for which I am unable to prove an inflammatory cause.

The question first arises, what is a tumor, and what is not a tumor?

In the sense of Virchow, any circumscribed elevation over a given surface, or any excessive enlargement, is considered a tumor. The products of specific inflammation, such as tubercle, gumma, glanders, lupus, and lepra, also the cysts and most of the monstrosities and the hypertrophies, would consequently belong here.

I consider the following neoplasms which are composed of new formed or overgrown tissues as *true* tumors :—

Fibroma.

Lipoma.

Chondroma.

Osteoma.

Leio-myoma.

Myxoma.

Lymphoma.

Sarcoma.

Glioma.

Papilloma.

Simple epithelioma (as represented by corns, horns, onychoma, etc.).

Carcinoma.

Tyroma (tubercular tumor).

Gumma.

Lupus, lepra, and glanders.

The following congenital neoplasms I consider as simple anomalies or *false* tumors :—

Angeioma.

Lymphangeioma.

Some keloids and other nævi.

Rhabdo-myoma.

Adenoma.

Dermoids and

Other cysts.

All these last-named neoplasms should be excluded from the tumors.

I suggest this, not because I cannot directly prove their cause to be inflammation, but because they are simple anomalies or malformations, just like a supernumerary finger. Nobody can acquire any of them, except the ordinary cyst. The individual must be born with them. Cohnheim calls them properly "monstrosities."

It is, however, possible that even here inflammation is concerned to some extent. Smallpox and syphilis, which are inflammations, are known to affect the fœtus in utero. Why, then, could we not have tumors as pre-natal inflammatory products? Still, I do not want to base my arguments on hypotheses. The fact is that children are born with large or small masses of any one of the above-mentioned congenital neoplasms.

Objection might be raised to the inclusion of adenoma in this category; but I have here reference mainly to the heterotopic adenoma, the perfect homologue of the mammae, which becomes prominent only at puberty, simultaneous with them, and is governed in time and grows by the same laws. The glandular acini which gave rise to the adenoma, and those from which the mammary glands started, were both deposited in the fœtus, and in both were dormant up to puberty, when they developed to structures perfectly alike, the difference being only that the one has its physiological purpose and location, and the other not. The histological distinction between them as given by authors I have not been able to see, after having examined *every* part of the structure. The homotopic adenomata, as occurring in connection with glands, are simple hypertrophies of any one of the racemose glands, or of a part of one.

The dermoid cysts are the best representatives of this group of anomalies. It is well established that they are simple local invaginations and misplacements mainly of epiblastic formations during early fœtal life. Certain parts of organs, such as skin, hairs, glands, teeth, etc., which usually are represented in these anomalies, proceed to full development and size, and no further. There is nothing pathological in these structures, except the location, unless combined with some other new formation.

Ordinary cystic formations are also frequently met with in the fœtus, although many cysts are acquired in later life by the agency of various pathological factors, including inflammation. Frequently tumors are the seat of cystic formation due to degeneration or softening in their interior. The formation of cysts is nearly always a passive process. Many arise from mechanical obstruction of outlets

of glands, or from the exudation of liquid into closed cavities. None of them has anything in common with tumors, except the tumefaction.

Angeioma and lymphangeioma and the keloids are exclusively congenital formations; they even seldom present themselves as tumefactions, and only then if subject to cavernous change and in combination with other lesions. There is no reason why these anomalies should be classed with the tumors.

The same may be said concerning rhabdo-myoma, the strictly congenital rare new growth, made up of misplaced striated muscular tissue.

It is in these congenital neoplasms alone that an inflammatory origin is not clearly evident.

OTHER VIEWS.

Without entering into details, I will at this point enumerate the other theories of the etiology of tumors.¹

These theories, although held by high authorities, and ingenious as they are, hardly go beyond the level of pure hypothesis. Hypotheses and speculations are easily disposed of by facts like those presented in favor of an inflammatory origin of tumors.

No tumor has ever been *proven* to have originated *spontaneously*, or to have been produced by a certain *dyscrasia* of the blood, or by *nervous influences*. We are no more justified in applying this or that pet hypothesis for the etiology of tumors without proving it, than in declaring a house to have arisen through the instrumentality of mysterious forces because we do not know who built it, and do not care to inquire by whom and how it was built.

The evolution and involution of tissues as conditioned by age, referred to by Thiersch and Rindfleisch, and best explained by Dr. C. B. Nancrede (in his highly suggestive communication to the Pathological Society, 1876), are regarded as important predisposing factors in tumor-formation, which at the same time decide the variety of tumors.

There can be no doubt that evolution and involution of the tissues influence the kind of tumor-formation; but I do not believe that these conditions in themselves predispose particularly to tumors, even in the presence of an over-supply of blood. We want certain changes in the integrity of the tissue (to be referred to later), and these can be brought about by the inflammatory process alone,—by nothing else.

¹ In my monograph, "The Etiology of Tumors," I have given all these views in full.

Due credit must also be given to Cohnheim for his embryonal theory of tumor-formation. Cohnheim uses the well-established congenital derivation of the dermoids, rhabdo-myoma, angioma, etc., as a basis for his hypothesis, and jumps at once to the conclusion that all tumors are congenital, and of embryonal origin. Some new formations which he admits to be of inflammatory origin—viz., gumma, tubercle, lupus, neuroma, osteophytes, etc.,—he excludes from the category of true tumors.

But how great a reduction in number will Prof. Cohnheim's list of true tumors experience should it be proven that all tumors are of inflammatory origin, save those few congenital formations which I have excluded from the list of true tumors!

Epstein (*Zeitschr. f. Heilkunde*, i., 1880) believes he has found anatomical proof of Cohnheim's hypothesis. He observed epithelial pearls in the mucous membrane of the gums, tongue, and genitals of new-born infants, and regards them as the famous supernumerary embryonic collections of cells. This, I think, is a great error. It has been shown by several observers that wherever squamous stratified epithelium exists, epithelial pearls may be found,—viz., in the epidermis, and in all epiblastic mucous membranes. I believe that the arrangement of epithelium in pearls is always a sign of retrograde change, and, as well as the arrangement of any kind of cells into nodes, signifies usually an ante-mortem act of cells, and not "dormant supernumerary embryonal collections."

To find further proofs for the embryonal theory of Cohnheim his pupils made extensive experiments. They succeeded; and the successful results, which were supposed to give a firm basis to the embryonal theory, were announced to the world in the renowned *Archives of Virchow*.

Unfortunately, however, for the Leipzig laboratory, the trifling efforts of American workers in experimental pathology have given results entirely opposed to those obtained by Cohnheim's pupils, and have probably forever demolished the beautiful embryonal theory of the etiology of tumors, as will be shown further on.

EXPERIMENTS.

Allow me now to refer one moment to the results of the experiments made with the object of ascertaining the cause of tumors. I will review here only the main points of interest; the details are given in my monograph.

Thus far but little success has been obtained in this line by experiments. Still, this much can be ascertained from them :—

First, that tumors cannot be inoculated by virtue of any infective or specific properties ; and

Second, that small living particles of tumors can be successfully transplanted from man to animals, and, upon transplantation, may continue to grow.

No one of the experimenters has really succeeded in producing a tumor by “inoculating” with tumor-juices, except in a very few doubtful instances.

The juices as occurring in malignant tumors are always the products of degeneration of the tissue or cells composing them. The cells suspended in that juice are dead, having undergone fatty or some other degeneration, and this is the reason that injection with juices fails. If particles of living tumor-tissue happen to be suspended in that juice, “inoculation” might succeed, but not with mere pure tumor-juice. In the few apparently successful inoculations with juices, particles of perfect tumor-tissue undoubtedly were injected together with them.

The notion of a specific tumor-virus held by some of the highest authorities is thus fully disposed of.

There are recorded a number of successful transplantations of tumors: *i. e.*, small fragments of tumors when put into the subcutaneous tissue of animals grew and enlarged in size as long as observed, if the conditions were favorable.

This, however, does not prove anything for the etiology of primary tumors. It has been shown that a cock’s spur, if it be transplanted from the leg to the comb, will often grow excessively ; we have another most perfect parallel to transplantations of tumors in skin-grafting and in plastic surgery.

Other observers, again, impressed with Cohnheim’s idea that tumors arise only from misplaced (heterotopic) cells or tissues, experimented as follows :—

1st. Particles of tissues taken from *adult* animals were introduced into the circulation and into the interior of organs, but they failed to grow, and were ultimately absorbed.

2d. Fœtal tissues (particles of embryonal cartilage, etc.) were similarly transplanted, and they grew and developed to moderate-sized tissue-masses (tumors).

Through these results Cohnheim’s proposition that tumors arise only from misplaced *embryonal* cells was regarded as proven.

But this was too hasty a conclusion, and it appears also that those experiments were conducted very carelessly, as the results could not be confirmed.

The exhaustive experiments which Henry Wile made in the pathological laboratory of the University of Pennsylvania (partly quoted in my monograph, and partly not published yet) positively prove that transplanted *adult* tissues grow as well as foetal ones, and never became absorbed in carefully executed experiments. Thus the much-dwelt-upon proofs for the embryonal hypothesis of Cohnheim are gone.

INFLAMMATION AS THE SUPREME CAUSE.

Allow me now to review the arguments in favor of, and the proofs for, an inflammatory origin of tumors, as brought forward in my paper. These are of three kinds: 1st, proofs by analogy; 2d, clinical and statistical proofs; and 3d, microscopical proofs.

1. *The close analogy of tumors and inflammatory products is strongly in favor of our proposition.*

Careful study and comparison have shown that no line of distinction can be drawn between true tumors and chronic inflammatory products; in fact, many of the latter are recognized as true tumors.

The criterion of true tumors is regarded to be their tendency to permanency in contradistinction to inflammatory products, which tend to disappear. The cases collected and the views of reliable observers recorded in my monograph show this to be incorrect. It has been proven that tumors occasionally heal and disappear. On the other hand, it is well known that only acute inflammatory products tend to disappear, while many chronic ones never do disappear, and that the symptoms and cause of the latter are frequently less obvious than in the case of tumors.

The connective tissue which, in proliferating, constitutes the main bulk of elephantiasis and of the cirrhosis of organs and a good many other pathological tissues outside of tumors, never disappears.

Virchow properly considers elephantiasis Arabum and soft fibroma morphologically and etiologically identical, and in the same sense he does not admit any difference between the connective tissue of an advanced cirrhosis of organs and that of a diffused fibroma. In fact, we are only in the habit of calling a proliferation of connective tissue in the mamma an intercanalicular fibroma, because the connective tissue affects an external part, while a similar affection of the liver or kidney

we term an inflammatory one—a cirrhosis. Why should we make such a distinction?

Gummata, tubercles (tyromata), lupus, the well-established products of inflammation, are unquestionably true tumors.

Lucke observed that sarcomata in young individuals occasionally grow as rapidly as acute abscesses, and that they have been frequently mistaken for the latter.

Tissues which are most liable to be the seat of inflammation are also the most *common seat* of tumors. Again, those tissues which do not participate in active inflammatory processes (ganglionic and striated muscular tissue) seldom or never give rise to tumors.

The extensive and careful statistics of Dr. D'Espine, of Geneva, show that the os uteri and the stomach are the most frequent seats of primary cancer, and they are also distinguished for their remarkable liability to catarrhs. Virchow has repeatedly pointed out in a catarrhally inflamed gastric mucous membrane the gradual transition to carcinoma, a fact observed also by Dr. J. H. Musser and myself.

The healing process in malignant tumors (wherever it occurs) is precisely the same as that of an ordinary granulating ulcer. Here and there, healing is accomplished by the additional formation of connective tissue,—*i. e.*, cicatrization.

But the most beautiful analogy between tumors and inflammatory products is demonstrable by the microscope, which led to the discovery of new and important facts.

2. *Clinical and statistical proofs.*

My own experience is limited, but in the cases of tumors in which I had the opportunity to get the history myself, or where I insisted upon an exhaustive anamnesis in the cases of others, it was possible in nearly one-half to trace out a local inflammatory process preceding the tumors at some time or other. Sometimes it dated years back. Careful inquiries nearly always revealed some cause,—*viz.*, an injury, long-standing irritation, mechanical or toxic, or an impaired or excessive use of the part, pressure, or a long-standing catarrh, or something of that nature.

It is also an established fact that those organs and regions of the body which, from their position and their function, are most exposed to injuries or irritation are the most usual seat of tumors. This is proven for the orifices of the digestive and genito-urinary tract, which are so much exposed to injuries and are also classical seats of especially malignant tumors.

Primary carcinoma of the gall-bladder has been repeatedly traced to gall-stones; that of the urinary bladder to a similar cause.

For surface-carcinomata an inflammatory origin may safely be regarded as proven. I know of scores of epitheliomata which had been traced to little sores produced by injury. Nearly all those everlasting leg ulcers are epitheliomata.

It is just here that the influence of evolution and involution of tissue upon the variety of tumor does not hold good. Repeatedly have I seen epitheliomata of the lower extremities in young persons directly produced by injuries, burns, etc. (Clinical service of Prof. Agnew.)

Who will deny the inflammatory origin of the very common epithelioma of lip? or that of the tongue or penis?—the first nearly exclusively occurring in smokers, the second always being associated with injury of the tongue by sharp teeth or otherwise, and the third with congenital or acquired phimosis.

Chronic inflammations of the skin, as occurring among workers in coal-tar and paraffin manufactories, etc., commonly lead to epithelioma. The chimney-sweeper's carcinoma has a similar origin.

It has been proven that long-continued catarrhs of the stomach (particularly in drunkards) lead to carcinoma.

Through irritation and injury common warts and scars are produced; further repeated injuries very frequently convert them into malignant tumors (carcinomata, sarcomata).

Prof. Agnew removed from the back of a middle-aged person a sarcoma developed secondarily in a scar. Some time previously he had removed (from the same patient) from the same spot, or from above that spot, a lipoma.

Sarcomata are commonly due to direct injury; neuromata exclusively so.

Glioma and tyroma, as met with in the brain, are nearly always traceable to falls and blows.

Hundreds of cases of fibroma, lipoma, chondroma, and osteoma have been traced by distinct and clear histories to pressure and irritation, or directly to blows, fractures, cuts, and other injuries.

Winkel, who investigated exhaustively the etiology of fibromata and myomata of the uterus, came to the conclusion that these tumors are caused directly either by excitants, viz., coition, injury, abortion, rough removal of the placenta, cellulitis, or, indirectly, through repeated lifting, shock, sudden hyperæmia, etc.

No reliable line of distinction can be drawn between the lymphomata and lymphadenitis.

Any one can convince himself of the above-mentioned facts by looking carefully over the literature, and by taking careful histories of his own cases. Hundreds of tumor cases of positively traumatic origin are also recorded in the classical works on tumors of Virchow, Weber, Müller, and Broca.

Unfortunately, however, these facts are not generally known; the literature is not sufficiently studied, and the histories of tumor cases are not sufficiently carefully inquired into.

Inflammation is the only factor which has been traced to be the positive cause of tumors in a number of cases. This is proven by high authority and statistics. But, as these authenticated cases of inflammatory origin are in moderate number, and as those with no cause (by reason of careless note-taking) are in enormous majority, the inference is drawn that inflammation has little or no significance in the pathogenesis of tumors.

I beg leave to argue as follows: In a certain number of cases it is positively known that inflammation preceded and was the cause of the new growth. In regard to the remaining cases of tumors we know nothing; no positive cause could be traced. Hence I think it logical, for the present, to consider inflammation as the cause of all true tumors. All other alleged causes are only speculations, and nothing reasonable can be brought forward against the inflammatory theory. Speculations are valueless, I think, in the presence of positive facts, even if these be few in number. In science any number of negative results are always disregarded in the presence of even a few positive facts. *Until proof to the contrary be given, we are at present, by a mass of evidence, forced to the conclusion that tumors represent merely one of the terminations of inflammation.*

3. *Microscopic proofs.*—Here I will make the following abstract from my first paper:—

The question now arises, in what way does inflammation produce a tumor, and why and when does a tumor develop after an injury? Why is not every injury followed by a tumor, if inflammation is the cause? Prof. Maas's¹ ingenious answer was that it depends upon the presence or absence of Cohnheim's supernumerary embryonic cells at the seat of the injury. If those misplaced or aberrant cells happen to be present

¹ Berliner Klin. Wochenschrift, No. 47, 1880.

in a part, a trauma will induce inflammation, followed by a tumor; if no extra cells are present, a simple inflammation will follow, and nothing more. But this is only an hypothesis; it cannot be demonstrated. Embryonal (fœtal) cells could not continue to exist unchanged in the adult individual; nor do they need to be pre-existing in order to form a tumor. They can be and are always created by any inflammatory process.

I will try to answer the above question by facts which microscopic examination reveals, and which will show that the study of histogenesis must go hand in hand with that of the etiology and possibly may disclose the mysteries of the cause of tumors.

It is true that direct observation of active pathological processes cannot always be made. In the case of tumors, only inferences of previous cell-activity can be drawn from the microscopic picture; but the pathological process can frequently be traced out under the microscope, from the various transitional stages of the elements of the new forming or formed tissue.

It is in accordance with the modern views to say that every tumor has its strict physiological prototype. Even for the carcinomata, only the peculiar atypical arrangement of the cells remains a criterion, while the cells themselves are supposed to be strictly identical with those found normally.

It appears to me, and the more I study the histology of tumors the more I become convinced, that any variety of cells composing a tumor are not identical with those found normally, but resemble those met with in chronic inflammatory products.

In tumors, the shape and the peculiar variations in size of the cells and nuclei, the character of the intracellular network and of the amoeboid motion of certain cells, the intercellular substance, the occasional arrangement into nodes, the relation to reticulum and bloodvessels, and the peculiarity of the latter, are all precisely like what is found in chronic inflammatory products and not like normal tissues.

There is a great difference between the tissue-elements of a fibroma and those of normal connective tissue, for example.

I shall give briefly the details of my investigation of the structure of fibromata, which, when completed, will be published and illustrated elsewhere.

Concerning the structure of normal connective tissue, the following seems to be generally established and in good preparations quite demonstrable :—

The ultimate connective-tissue fibrils (the fibrillar variety) are in varying number united together to form bundles; these again occasionally unite to form larger bundles; these bundles arrange themselves at different localities in various manners, *i. e.*, parallel as in tendons, or as a lattice-work in membranes, or decussate at different angles and in all possible directions in all other localities, leaving between small spaces, these spaces being dependent for their shape and size upon the arrangement of the bundles. They communicate with one another, and thus form a system of channels throughout the whole connective-tissue system of the body. These channels contain a small amount of fluid containing *mucin*, and they are the receptaculi of the sometimes enormous quantities of serum in œdema. These same spaces or channels may also get filled with air, producing emphysema beneath the skin and other parts of the body.¹

Von Recklinghausen has shown that the spaces in the connective-tissue communicate with the lymphatics, and he calls the spaces juice-channels; they act as “*vasa serosa*” (Orth), conducting the serum from the bloodvessels to the lymphatics, and “*feeding*” (Tyson) the tissues.

By the nitrate of silver method of Von Recklinghausen, which is now the common property of all the laboratories of the world, it can be easily demonstrated that each of the connective-tissue bundles spoken of is surrounded by a distinct membrane composed of large flat cells. These flat, so-called endothelial cells are very thin, nucleated, and are closely united at their periphery with one another, so as to form continuous membranes or sheaths, which envelop each or several fibrillar bundles and thus at the same time form a lining for the spaces between them. Without nitrate of silver the endothelial cells cannot be seen; all that is seen are the nuclei of the cells, round or oval in shape if viewed from above, or spindle-shaped if the whole cell is seen in profile. I will not enter into further details here; this suffices to make myself now intelligible concerning some points in the histology of connective-tissue tumors, particularly fibromata.

I investigated by the nitrate of silver method three specimens of fibroma: 1st, a small, hard fibroma from the finger of a girl, æt. 20, developed from the tendon; 2d, one of the size of two fists from the

¹ The subcutaneous tissue of the whole body can be filled with air, so as to produce enormous emphysematous disfiguration, by forcing air through blow-pipes at a few points or possibly even from only one point of the body below the skin. I have seen children purposely prepared in this way for beggars' purposes.

broad ligament of a woman, æt. 35; and 3d, an intra-uterine fibroma of the size of one fist from a woman, æt. 40.

I might say at the outset that in the preparation of the first and third specimens I failed altogether to discover any perfect endothelial sheaths surrounding the bundles of fibres, which were so beautifully seen in a preparation of tendon made for comparison simultaneously with the fibroma specimens. In specimen second only a few perfect endothelial sheaths were visible. The microscopic picture of one of the silver preparations (from specimen No. 1) was this. The fibrils were on the average much thicker than in normal connective-tissue; some running straight, others rather wavy and not quite parallel with one another, frequently decussating. Only a few perfect fasciculi or bundles of fibres were seen, but most of them had not a trace of endothelial ensheathment. Some had a partial endothelial sheath in some places, and here the bundles appeared constricted. In several places were seen irregular protoplasmic masses apparently in connection with the fasciculi and proved to be partially detached endothelial cells. Between the bundles were seen several groups of young indifferent cells, resembling white blood-corpuscles. Other cells were double the size of the latter, some spindle-shaped and with prominent nuclei. The latter were seen occasionally in a state of division or were already divided. They resembled remarkably the germinating endothelial cells from serous surfaces, as described by E. Klein, of London, represented by him in his *Atlas of Histology*, Plate VI.

I interpret the microscopic picture as a whole thus. The endothelial cells composing the sheaths of bundles of connective tissue have become isolated, and hence the sheaths are destroyed. The boundaries being removed, the liberated connective-tissue elements grow with great vigor. The growth is perhaps promoted yet more by the presence of the serum of the juice-channels, with which the cellular and fibrillar elements now come in direct contact, the sheaths being destroyed. The cells and fibres here, as in elephantiasis, "feed" (as Prof. Tyson would say) upon that serum in which they are soaking. The endothelium is proliferating (germinating, *Klein*), and probably gives rise to those groups of indifferent cells which evidently form the main source of the new growth. Foerster¹ has pointed out that in the development of fibromata the fibres arrange themselves more or less concentrically around and develop from these islands of cells, thus

¹ *Atlas der mikroskopischen und pathologischen Anatomie*, Leipzig, 1855.

giving rise to the lobulated appearance of this new growth. It is also very probable that emigrated white blood-corpuscles assist in forming these collections of cells.

What interests us at present, however, is the absence of the endothelial sheaths in the connective-tissue bundles in fibromata, and that a fibroma has this feature in common with all connective-tissue formations which owe their origin to inflammation will be shown directly.

I can affirm the absence of endothelial sheaths in the newly-formed fibrillar connective-tissue as met with in cirrhosis of organs which invariably accompanies the proliferation of the alveolar connective tissue in such situations. It would be very desirable that other histologists would undertake research in this direction.

Cornil and Ranvier¹ describe the disappearance of the endothelial ensheathments in connective tissue which is the seat of inflammation. They describe the appearance as follows: "The fasciculi are smaller; less distinctly fibrillar; they do not appear to be enveloped by a special layer which limits them and which causes them to swell irregularly when acted upon by acetic acid." They consider that the "large flat cells" are replaced by embryonic tissue.

The inflammatory process is, to my knowledge, the only factor which can disconnect or isolate endothelial or epithelial cells united together to form a certain lining or covering. Let us take, as an instance, the lung. The flat cells which form the lining of the air-vesicles are so closely united or grown together in the normal adult individual that no means at our command at present can isolate them. But in catarrhal pneumonia the inflammatory process demolishes that lining instantly; the cells which compose it "return to their embryonic state" (Stricker), and they become completely isolated.

The abnormal increase in bulk of tissue in both the fibroma and the inflammatory connective-tissue products, appear to me to be due to the same causes:—

1. The removal of the boundaries which keep the fibres intact, viz., the destruction of the endothelial ensheathments.
2. The proliferation of the endothelial cells of these destroyed sheaths and of the connective-tissue elements themselves, and probably in addition, white blood-corpuscles.

¹ A Manual of Pathological Histology, translated by Shakespeare and Simes, Philadelphia, 1880.

If the endothelial sheaths of the connective-tissue bundles and other normal boundaries are re-established in the inflamed tissue, then it will return to its normal state, or in case of loss of substance will heal by permanent scar-tissue. The healing process is then perfect.

On the other hand, the same tissue will give rise to a fibroma if this healing process is imperfect; *i. e.*, the endothelial ensheathments are not re-established, the connective-tissue elements remaining freed from any restriction proliferate of their own accord, grow above the physiological limit, and thus inflammation terminates in a tumor.

Hence, from histogenetic grounds, I would suggest that *fibromata should be classed as a product or rather as one of the terminations of inflammation.*

This is also in accord with clinical experience.

Now, is an inflammatory origin less evident in other tumors? Can there be shown any positive microscopic difference, for instance, between a mass of inflammatory granulation tissue and a sarcoma? It cannot. To my knowledge, distinguished histologists have repeatedly had sad experience of this.

If the discoveries of Classen and Woodward should prove correct, we shall, to my mind, have an additional proof that cancer is only one of the terminations of inflammation. I will quote the following:—

Woodward¹ says, "My own studies of thin sections led me to the conclusion that the migration of white blood-corpuscle played a great rôle in the development of cancerous growths, and that at least in certain cases the cancer cylinders were formed by the transformation of these corpuscles, which first accumulated in the lymphatic capillaries and the passages leading to them."

Classen² is still more positive, saying that he has proven "that the cells of cancer cylinders and all the elements of cancerous growths are no other than migrated white blood-corpuscles escaped from the bloodvessels."

Though in my own research I have not succeeded as yet in confirming the observations of Woodward and Classen, they are possibly correct, and I utilize them as coming from such high authority. Besides, they correspond remarkably with what I believe I have established for fibromata.

¹ The Structure of Cancerous Tumors. Toner Lectures, Washington, 1873.

² Ueber Cancroid der Cornea, etc., Virchow's Archiv, vol. 1., 1870.

My view of the histogenesis of fibromata holds good also for primary glandular carcinoma.

The glandilemma or basement membrane in glands (wherever such exists), upon which the epithelial cells rest, may be destroyed in precisely the same manner as the endothelial sheaths of the fibrillar bundles. This is demonstrable in carcinoma beginning to develop in a gland, or in the transformation of an adenoma into a carcinoma. Here, as in a fibroma, only an inflammatory process can accomplish this destruction of the normal boundaries. These boundaries, if not re-established after an injury by perfect healing, leave nothing to prevent the epithelial cells from travelling into the surrounding connective-tissue spaces and thus to form a carcinoma.

I have here reference to the destruction of the endothelial boundaries which form the basement membrane of the epithelium alone. The endothelial ensheathment of the connective-tissue alveoli remains perfect in the carcinomata unless it becomes inflamed.

It is not the want of resistance of the surrounding tissue (as is generally held), but simply the getting loose of the normal cells from their place of attachment, which constitutes the formation of a malignant tumor.

It is the mobility of the cells, I think, that conditions the malignancy of a tumor. Any tumor, even the most benign one, would be eminently malignant if the cells composing it could get loose and travel through the widely open paths of the system of juice-channels.

In benign tumors the cells are more or less fixed, hence no metastasis. The endothelial basement membranes and ensheathments are, however, here also defective. The physiological boundaries which maintain the equilibrium and keep the cells in position and in harmony with one another are found absent in that tissue which gave rise to the tumor-formation.

As it is not proven so far that any other pathological process besides inflammation is capable of destroying the endothelial ensheathments and basement membranes, I am driven to the conclusion that all true tumors are direct products of the inflammatory process, and that true tumors should be considered as one of the terminations of inflammation.

Dr. S. W. Gross said that the main propositions propounded by Dr. Formad were, first, that all tumors are the products of the inflammatory process, and, secondly, that in the development of tumors there is a destruction of normal boundaries. Gumma and tubercle are regarded by the author as tumors; but Dr. Gross thought that term

should not be applied to the temporary products of specific inflammations, but that it should be restricted to permanent additions to the normal tissues. Dermoid cysts ought certainly to be included, as should also angeioma and lymphangeioma. The former naturally comes under the classification of cystic growths, while the latter—in regard to the causation of which Dr. Formad confesses that he has strained a point—are not merely congenital enlargements of pre-existing vessels, and should, therefore, be retained among the neoplasms. While it is true that in carcinoma of the breast the membrana limitans, or glandilemma, of the acini and ducts is destroyed, it is equally true that it remains intact in adenoma of that organ; so that, in the formation of tumors, normal boundaries are by no means always destroyed. Dr. Gross was convinced that inflammation, or a process nearly related to it, plays an important part in the etiology of many tumors, but he thought that Dr. Formad was too exclusive in his theory. He, moreover, believed that Dr. Formad was too sweeping in his assertion that inoculation with the juice of neoplasms was incapable of begetting similar growths. Dr. Formad, indeed, quotes several experiments which disprove his own positive assertions; and Dr. Gross related the following cases, which he thought supported the doctrine of the inoculability of tumor-juices.

The first case shows that sarcoma may be transmitted to man from an animal, and the second and third demonstrate infection in the same individual other than by metastasis.

CASE I. An ox was affected with a subcutaneous tumor behind the scapula, which proved, on removal, to be a medullary sarcoma. A few days before the operation the owner made an incision into the swelling on account of pseudo-fluctuation, and there was a sanious discharge for some days. The wife, æt. 23 years, was in the habit of cleansing the part, and had at the time a small wound on the outer side of the fourth finger of the right hand. In a few days a small warty excrescence was noticed on the finger, which soon became the seat of burning pain, and attained a diameter of fifteen millimetres in a month. It was covered by a whitish-gray pellicle, and Dr. Kuhn, of Niederbronn, found it to be a medullary sarcoma. (*Magazin für die Gesamte Thierheilkunde*, 1862, p. 328.)

CASES II. and III. Dr. Reinecke, of Hamburg, has recorded two examples of the inoculation of the canal formed in tapping the abdomen in carcinomatous peritonitis. In both, the primary disease was carcinoma of the ovary, with secondary affection of the mesenteric

glands and the peritoneum, resulting in ascites, for which paracentesis was performed, five times in the first case and twice in the second. In both instances carcinomatous nodules appeared in the track left by the trocar, which, on post-mortem inspection, were not found to be continuous with the carcinomatous peritoneum, but separated from it by a layer of sound tissue. (*Virchow's Archiv*, Bd. 1.)

Dr. Tyson thought that whatever else might be disputable as to Dr. Formad's view of the etiology of tumors, he was correct in saying that there were more facts in support of the inflammatory view than could be adduced by the advocates of other theories. This much he was willing to concede, but still thought the proposition not proven. The dyscrasia theory has been practically disproved by Virchow. The spontaneous theory has some points in its favor, and it cannot be *disproved*, although, on the other hand, it has fewer facts in its favor than has the inflammatory view. Cohnheim's theory has nothing in its favor beyond the occurrence of rhabdo-myomata, dermoid cysts, angiomas, and lymph-angiomas, etc., which are allied congenital growths. The inflammatory view has two sets of facts in its support,—viz., the occasional operation of causes which are identical with those which produce inflammation, and the histological resemblance presented by certain tumors, as fibromata, to the products of inflammation as seen in cicatrices, and, if Dr. Formad's last observation is correct, the further histological similarity as to the absence of the limiting endothelial membrane surrounding the connective-tissue bundles. Dr. Tyson agreed with Woodward and others in thinking that the time had not yet come for a satisfactory determination of the etiology of tumors. Certain facts adduced by Dr. Formad have not the weight that he supposes,—viz., the want of permanence of tumors and the persistence of inflammatory products. The instances of both related are but exceptions to the rule. On the other hand, however close may be the resemblance of the histological elements of some tumors to those of inflammation, there are many others in which no such resemblance exists; such is the fact with regard to the carcinomata and many histoid tumors, as the chondromata and osteomata particularly. The attempt, however partially successful, is nowhere paralleled in inflammatory processes.

As to the position to be accorded to such formations as angioma, lymphangioma, and dermoid cysts, Dr. Tyson fully agreed with Dr. Formad that, accurately speaking, they have no place among tumors. We continue to place them there rather from force of habit than for

any scientific reason. Only in one particular—their correspondence with the etymological definition of “tumor,” which means literally a “swelling”—do they comport with the correct notion of tumor.

Dr. F. P. Henry said that he could not accept the theory of the inflammatory origin of tumors except in the general sense that they, as well as inflammatory products, are the result of perversions of nutrition. Its acceptance would necessitate a change in our views regarding the inflammatory process, compelling us to speak of a fibromatous, a lipomatous, an enchondromatous, and other hitherto unheard-of forms of inflammation.

While facts such as those mentioned by Dr. Formad furnished strong evidence in favor of the inflammatory origin of tumors, it should not be overlooked that there are at least equally strong facts opposed to this theory. Chief among these were the extraordinary frequency of inflammation and the comparatively extreme rarity of tumors. If a direct causal connection existed between inflammation and tumors, the latter would be more frequent. Dr. Formad had quoted the statement of a United States military surgeon that certain tribes of Indians enjoy an almost complete immunity from tumors, and there is no doubt whatever that the mode of life of these same Indians must render them peculiarly subject to inflammatory affections.

Prof. Tyson had referred, by way of illustrating a point in favor of the inflammatory theory, to the likeness presented by a fibroma to a mature cicatrix, and that presented by a sarcoma to granulation tissue. These are mere coincidences. To make this illustration of value it should be proved that the fibroma originates as a sarcoma, which, it is scarcely necessary to say, cannot be done.

Dr. Henry acknowledged the pleasure and profit he had derived from Dr. Formad's pamphlet, and expressed his belief that it would be regarded as a standard work of reference by those interested in the subject of the etiology of tumors.

Dr. Nancrede said that in reading Dr. Formad's valuable paper his attention had been arrested by certain statements from which he could not but feel compelled to dissent. According to the commonly-accepted view of the process of ossification, the discovery of islets of cartilage in adult bones is precisely what one would expect, especially when we know that traces of chondrigen are found in analyses of mature portions of the skeleton. Instead of being “misplaced germs,” they are merely remains of the calcified fetal cartilage situated at the points of mutual intersection of the periosteal ingrowths, which finally sub-

stitute all except traces of the fetal structure. Even accepting Cornil and Ranvier's view,—which the speaker thought was, after all, reconcilable with the observations of other authors,—the “misplaced-germ” theory was utterly untenable. Dr. Nancrede thought that Dr. Formad had misunderstood his views as set forth in the quotation from his paper, as he would rank himself among the “inflammatory” as well as the “spontaneous” theorists. The speaker then gave a *résumé* of his own views, supporting them by certain positively ascertained facts as to the condition of the mammary gland at various ages, the effect of varying blood-supply to it and other organs, etc. He then stated that he considered Dr. Formad's views were incorrect as to “natural healing,” or the reverse in its causative relation to morbid growths. Dr. Nancrede propounded the following: that when the irritant and the condition of the tissues were so related that the proliferation of cells was such as to keep pace with a sufficient blood-supply to admit of their development into tissue, normal healing occurred. If this proper relation failed to obtain, suppuration, caseation, or, perhaps, under certain circumstances, various morbid growths, would result. The speaker then mentioned certain facts which could be actually proven as to the relative atrophy of the connective tissue of the lip, the effect of continuous local irritation on the development of epithelioma, certain well-attested physiological facts, and contended that the missing links in his chain of, not reasoning, but facts, were practically demonstrable. He therefore repudiated Dr. Formad's dictum that all views but the inflammatory were mere theories,—“*that where nothing is proved there is nothing to disprove*,”—and quoted from the lecturer's paper on page 46, where he contended that the conclusions were *purely* theoretical and not logically warranted. Dr. Nancrede then endeavored to show that the failure of the connective-tissue bundles in rehabilitating themselves with their endothelial investment, if confirmed, and specially if demonstrated as a weakening of the connective-tissue barrier against epithelial ingrowths, was merely due to want of equilibrium between the blood-supply of the two tissues.

Dr. Chas. K. Mills said that he wished to put on record, in connection with Dr. Formad's valuable paper, a few notes on ten cases of brain tumor in which the post-mortem examinations had been made by him. These were cases in which close inquiries were made as to probable causation. In the majority of them, as will be seen, a history of traumatism was given. The notes were with reference to the history and the nature of the growths, as follows:—

CASE I. Fall from high door-step, striking head. Fibroma.

CASE II. Wounded in the head by glancing bullet. Gumma.

CASE III. History of blows on the head and of syphilis. Gumma.

CASE IV. History of blows on the head and of syphilis. Gumma; also softening and abscess.

CASE V. Kicked by a horse on the head. Fibroma.

CASE VI. Thrown from a horse and kicked on the head. Two growths; fibroma and gumma.

CASE VII. History of syphilis. Gumma.

CASE VIII. No history. Glioma.

CASE IX. No history. Glioma.

CASE X. No history. Carcinoma.

Dr. E. O. Shakespeare felt that he could not allow the debate to close without expressing his high appreciation of the value, to the American physician, of the labor Dr. Formad had so successfully and learnedly performed in collating from the literature of the languages of the civilized world almost all of importance that has been thought and performed by distinguished men while attempting to elucidate the causes of tumors, and in classifying and abstracting briefly, clearly, and forcibly, the various opinions of investigators. He had listened, much interested, while the lecturer with great ability and ingenuity proceeded to unfold and support his own belief concerning the etiology of tumors, and he had given close attention to the progress of the subsequent debate. He confessed that he had made no great study of the subject in question, and therefore did not feel entitled to entertain or express any very positive opinions; yet, during the course of the reading of the paper and of the discussion which had followed it, he had become more and more convinced of the necessity of exercising great caution in the acceptance of assumptions which may have little for their justification beyond a quasi-sequential order of appearance of certain phenomena, which is often, but by no means always, recognized in the history of tumors. He very much doubted the possibility, in the present state of our knowledge, of proving that inflammation either was or was not the essential cause of tumors. Certainly the lecturer, as well as other experienced investigators, must be credited when he affirms that in the majority of cases of tumor in which an adequate history has been recorded the growth has been preceded by a local inflammation or an injury. And yet even in these cases (supposing, for the sake of argument, there were no other) what right has any one to assume that the previous inflammation has acted as any

other than a simple exciting cause? and who can rationally declare the tumor to be one of the natural terminations of the inflammation?

If the lecturer thinks he has discovered a general law concerning the etiology of tumors, let him and those who seem inclined to accept his hypothesis for one moment consider its application to syphilitic and tuberculous growths.

Dr. Mills has related, in the course of the debate, a number of cases of brain tumor, the histories of which showed that they followed a severe blow or other traumatism. In some instances the tumor proved to be sarcomatous, in others tuberculous, in others gummatous. Dr. Shakespeare referred to these particular cases because Dr. Mills had related them as perhaps offering some support to the hypothesis advanced in the paper, and had incidentally referred to him as personally cognizant of several of them. These cases are no more, perhaps no less, typical than others of that large class from which the essay has been made to deduce the general law enunciated this evening.

Will the lecturer take the position (seemingly absurd in the light of our present knowledge of syphilis and tuberculosis) that the tuberculous and the syphilitic tumors, no less than the sarcomata, are simply the natural terminations of an ordinary inflammation established by a traumatism? Or will he rather prefer to further curtail the list of tumors for the purpose of his theory, and erase the names of tubercle and gumma?

If the latter horn of the dilemma be elected, he would suggest the propriety of placing true carcinoma in a category very near to that of tubercle and gumma, for there are very many parallels and similarities in their clinical and pathological aspects.

He declared that he could see no cogent reason why some authors, in drawing the line of demarcation between abnormities which are and those which are not to be regarded as tumors, have placed upon the one side carcinomatous growths and upon the other side have ranged the permanent products of syphilis and tuberculosis. He did not recognize mere relative size as an adequate distinction between one morbid product which must, perforce, be a tumor, and another which must not be so classified. Carcinoma sometimes presents in its history the phase of miliary eruptions, and, on the other hand, tubercles oftentimes form a confluent tumor-mass of very considerable size and delimitation. All that is at present known of carcinomatosis and of tuberculosis would seem to warrant the belief that in both there is frequently a strong hereditary predisposition. In both, from the locus of primary

manifestation of disease the system may become infected by way of the lymphatics; in both the chain of lymphatic glands along the course of the lymph-vessels which lead from the primary growth may, and often does, form a cordon to prevent, at least for a time, the contamination of the general system; in both, when the morbid influence passes these natural barriers and reaches the circulating blood, metastases in various situations usually occur.

Notwithstanding the research and the observation and the ingenuity of the lecturer, Dr. Shakespeare thought that, as yet, we have no satisfactory reasons for attributing to an ordinary traumatic inflammation any agency in the development of a tumor beyond that of a simple exciting cause.

Dr. Formad, in closing the discussion, said, in reference to Dr. S. W. Gross's remarks, that he did not think it probable that tumors could arise from inoculation with tumor-juices unless the latter were the carriers of living tumor-particles. He believed that even in the three cases of apparent inoculation with tumor-juices just referred to by Dr. Gross there was no evidence at all that such small tumor-particles were not suspended in the juice, and did not effect the transplantation of the new formations.

In reference to Dr. Tyson's remarks that the similarity between inflammatory products and tumors was limited to only a few instances, Dr. Formad maintained that this similarity was applicable to the majority of tumors, and, contrary to Dr. Tyson's view, was easily demonstrable,—*e. g.*, in carcinoma. Dr. Formad dwelt upon the gradual transition of inflamed skin into a canceroid, and of a catarrhal inflammation of the stomach or gall-bladder into a carcinomatous growth,—the microscopic picture showing the direct merging of the primary inflammatory changes into carcinoma, and that it is impossible to point out where the one ends and the other commences.

Dr. Formad could not agree with Dr. Henry that there were necessary "a fibromatous, a lipomatous, an enchondromatous, and other unheard-of forms of inflammation" in accepting the view of an inflammatory origin of tumors. Dr. Formad thought that the ordinary process of interstitial and parenchymatous inflammation and the laws which govern the new formation of tissues are sufficient to explain the histogenesis of the various tumors. Fibromata, he thought, should be regarded as one of the products of chronic interstitial inflammation; lipoma is nothing else than a uniform fatty infiltration of a fibroma, and a myxoma a mucoid degeneration of the latter. Before we can

have adipose tissue we must have connective tissue; and probably all pathological mucous tissue has its origin in a mucoid degeneration of simple connective tissue. The direct transformation of fibrillar connective tissue into cartilage has been proven by several reliable observers. Thus a chondroma is formed. We have a parallel to this in the process of ossification and in the formation of osteomata, etc. Dr. Formad stated that he was acquainted with no real facts that could be brought forward against the view of an inflammatory origin of tumors.

Dr. Formad agreed with Dr. Nancrede that the quantity of the blood-supply conditioned the growth or the destruction of tissues, and determined frequently the variety of tumor-formation. He maintained, however, that only the inflammatory process was able to prepare a tissue anatomically, or to predispose it to tumor-development. The destruction of the endothelial boundaries, the main causative factor, cannot be brought about by irregularity of blood-supply. Continuous hyperæmia, Dr. Formad thought, may bring on—for instance, in the mammary gland—a homotopic adenoma, which is only a simple perverted epithelial hypertrophy, and not a true tumor. An injury to the elements of the skin, in the same mammary gland, will, under circumstances referred to, produce a surface epithelioma. An injury affecting the connective tissue of the gland will predispose to sarcoma (rapid effect) or to fibroma (slow effect), while the same cause operating upon the glandular elements proper (destroying the glandilemma) may induce a hard or a soft carcinoma.

In reference to Dr. Shakespeare's remarks, Dr. Formad stated that he did not mean to declare tumors to be a "natural" termination of inflammation, and that he was perfectly willing to call them a perverted termination of the inflammatory process, occurring only if the healing process is imperfect or retarded.

Tubercle and gumma Dr. Formad did not exclude from the category of tumors, and in them he thought that he had one of the best supports for an inflammatory origin of tumors. Dr. Formad did not think that the causes of the inflammation were here pertinent. In the case of tumor-formation it made no difference whether the operating cause of the inflammation was a specific poison, or a trauma, or anything else. The specific virus is not the cause of the gumma or tubercle, but is the cause of an inflammatory process, which in turn gives rise to the new formation. If the inflammatory changes are arrested, no tumor develops. The excellent series of cases of brain tumors referred to by

Dr. Mills, Dr. Formad thought, supported admirably the view propounded.

Dr. Formad expressed his gratitude for the interest taken in the paper by the President and by the members of the Society, and for the many suggestive points ventilated by the discussion.

October 27th, 1881.

4. *A case of spontaneous atrophy of a tumor.*

Presented by Dr. J. B. ROBERTS.

Dr. J. B. Roberts made some remarks on an interesting case of atrophy of a skin tumor, which was probably of a fibrous nature, and connected with a nerve. He had not observed this diminution in bulk himself, and had had to rely upon the patient's statements, which, however, he had no reason to doubt were perfectly correct. The patient received, seven or eight years previously, a wound in the axilla which exposed the brachial plexus. Since then he has suffered from general pains, hyperæsthesia of the hands and feet, slight unsteadiness of gait, and difficulty after sitting in assuming the erect posture. The tumor had appeared on the shoulder a short time after the accident, and was very painful on pressure. A few weeks before Dr. Roberts saw him, the tumor suddenly commenced to grow smaller, until now it is reduced to probably a sixth of its original bulk.

January 12th, 1882.

5. *Apparent atrophy of a tumor previously reported, explained by the further history of the case.*

Read by Dr. J. B. ROBERTS.

At the meeting of January 12, 1882, I reported a case of apparent atrophy of a tumor of the left shoulder. The published account of my remarks, found in the *Medical Times*, is erroneous in that the wound spoken of was received *seventeen or eighteen years* ago, and involved the *opposite* axilla. Moreover, the tumor did not appear until four or five months previous to the time of the meeting, which was seventeen or eighteen years after the injury. The general neu-

rotic tendencies of the patient were merely mentioned to show the character of the individual, and not because the growth was supposed to be connected directly with the wound, as would be inferred from the published statement.

Since the meeting, the explanation of the case has become easy, and, as I stated, I would detail the subsequent course of the growth. I now do so.

On January 25, 1882, he came to my office, saying the tumor was very painful, and resembled a boil. Examination showed a small fluctuating swelling, purplish-red in color, without any areola of inflammatory redness, and showing no tendency to point at the seat of the former tumor. It resembled and evidently was a subacute abscess of a lymphatic gland. Incision and the insertion of a small tent were followed by speedy cure.

The unusual location on the top and posterior aspect of the shoulder, the history of long duration without inflammatory symptoms, the absence of other enlarged glands, and the exquisite neuralgic pain on pressure, made me look upon it as a fibroma. It was evidently, however, a chronic inflammation of a lymphatic gland and its circumglandular tissue. After remaining quiet several months, the tumor gradually became reduced in volume by absorption of the inflammatory deposits, and then abscess occurred in the gland itself.

April 12th, 1882.

6. The trophic system as a factor in pathological processes.

Read by Dr. ROBERTS BARTHOLOW.

Not long since, it was the prevailing notion to refer most or all pathological actions to an influence derived from the nervous system. About that time, the results of Waller's studies, and the remarkable observations of Mitchell, Charcot, and others, had thrown a new and brilliant light on the nature of the trophic nervous system, and the morbid conditions resulting from its perturbation. Now, germs occupy the first place in professional attention. It is true there is yet enough, and more than enough, of that mechanical humoralism which finds in the state of the vaso-motor system—in the variations in the vascular supply—ample explanation of all kinds of structural changes.

Since it has been shown that a mere relaxation of the vessels exists in the inflamed area, and that the condition leading to stasis and its

consequences is purely local in the walls of the vessels, the active agency of the vaso-motor system in the process of inflammation must be denied. That certain parts of the nervous system have to do with the nutritive functions, and that a morbid state of these parts sets up structural alterations in distant but related organs, are facts also which must be maintained as ultimate and true. Waller first distinctly enunciated the proposition, that a nerve when separated from its trophic centre undergoes degeneration. Mitchell and his associates of this city, Charcot, Schiff, Erb, and many others, have shown that irritation of nerve trunks is followed by various changes in connected and dependent organs. It is useless, indeed, to be reaffirming so obvious a fact. Let me, however, as a groundwork of the statements to follow, give the anatomical data: Certain masses of gray matter appear to be the seat of the trophic influence. The large multipolar or caudate cells of the fourth layer of the gray matter, which constitute the psycho-motor centre of the hemispheres, apparently are the trophic organs or centres for the fibres of the pyramidal tracts and probably of the brain in general, whilst the corresponding cells of the anterior cornua supply the trophic influence to the efferent fibres of the spinal cord, and ultimately to the muscles and joints. On the other hand, the central and posterior portion of the gray matter of the spinal cord is related to the nutrition of the skin. The efferent nerve-fibres stand in a similar relation to the parts supplied by them, so that when they are subjected to irritation degenerative changes occur in the same way as if the trophic spinal centres were affected.

Such is the anatomical basis of the trophic system. Is the trophic a separate endowment of these parts of the nervous system, or are the so-called trophic changes due to a state of the vaso-motor system? The answer to this question involves a consideration of the forms of lesions consecutive to affections of the supposed trophic centres, and of the vaso-motor system respectively. It will be most convenient to consider the vaso-motor system first. Broadly, there are two pathological conditions by which we may find this system affected: by irritation and by destruction of function, and their consequences. Irritation induces contraction of vessels, diminished functional activity of organs, and a depressed state of the nutrition of parts. When destruction is effected, function ceases, the vessels dilate, and a state of hyperæmia ensues. The results of irritation and destruction of function continue indefinitely without further mischief, although injurious effects may follow trivial causes. As far as the vaso-motor system is itself con-

cerned, no changes of importance, no structural alteration follow mere irritation of its nerves or ganglia, or even destruction of them. A condition may be induced in either case in which great mischief is potential merely.

Beside the centres of vaso-motor innervation which exist in the medulla oblongata and in the cord, there are at various points in the arterial distribution local ganglia, which have the power to maintain the vascular tonus somewhat independently. Otherwise it would be impossible to explain the recovery of vascular tonus after the vaso-motor centres are destroyed, and that it does recover is a fact which has been abundantly demonstrated.

There are numerous anatomical and physiological data showing an intimate association of the vaso-motor with the trophic system. The vaso-motor centre in the medulla oblongata is connected with certain definite areas of vascular distribution in the gray matter of the hemispheres. This arrangement is called by Meynert a projection system. Irritation of the centre is followed by contraction of the vessels, and an anæmia of the associated vascular area. Now, as in the fourth layer of the cortex are situated the trophic cells, the importance of such an arrangement is obvious. Again, there are, according to Eulenberg and Landois, vaso-motor centres in the cortex—for destruction of such parts causes vaso-motor paresis and elevated temperature in the opposite extremities—facts which explain the presence or absence of temperature and other changes in the vessels of the paralyzed parts in hemiplegia.

The vaso-motor centres in the cord are connected with special vascular areas. Schiff, Brown-Séquard, and others have found that injuries of the pons and medulla caused hyperæmia and ecchymoses of the lungs, pleura, kidneys, and the mucous membrane of the intestinal canal. Bernard, as all the world knows, caused albuminuria by irritating the upper part, and glycosuria by irritating the lower part, of the floor of the fourth ventricle. When the lumbar spine is injured, extravasations of blood take place in the suprarenal bodies. Injuries involving the vaso-motor centres in the spine are followed by changes in the temperature, and contraction or dilatation of the vessels of the paralyzed parts. In all of the cases of irritation or destruction of the vaso-motor centres, if the mischief is limited to them, and does not include the trophic centres, the resulting vascular changes do not involve alteration of the proper structure of parts. Besides variations in temperature, there may be excess or deficiency of certain glandular

secretions, but no actual lesions of the gland elements. Obviously, the influence exerted by the vaso-motor innervation is quite inadequate to explain the changes which ensue when parts are separated from certain defined areas of gray matter.

When those areas of gray matter—distinctive in structure—supposed to have a trophic function are irritated or destroyed, results of a peculiar kind follow. When the fourth layer of cells in the gray matter of the hemispheres is injured, the fibres of the pyramidal tract undergo atrophic degeneration. Disease of the multipolar cells of the anterior cornua of the spinal cord causes not only paralysis, but rapid wasting and degeneration of the paralyzed parts, which exhibit Erb's phenomena of the reactions of degeneration. Various affections of the skin, falling out or abnormal growth of hair, deformities of the nails, pigmentation of the skin, erythematous and papulous eruptions, urticaria, herpes, ecthyma, etc., ulcerations, bed-sores, etc., are results of irritative lesions of the gray matter of the posterior columns, or about the posterior nerve-roots. Whether the trophic influence proceeding from these parts is transmitted by a nerve fibre having this special function, or by the motor or sensory fibres, is unknown; but it is probable that the trophic centres of the anterior cornua, and of the posterior nerve-roots, exercise their functions through the nerves proceeding from them respectively. That the trophic centres exist, there seems to be no possibility of doubt; that trophic fibres proceed from them having no other office or function, it is not necessary to affirm, for the motor and sensory nerves, which are subjected to the trophic influence, may readily take on the function. One of the two hypotheses is necessary, since ordinary motor and sensory nerves do not possess a trophic function.

What is the nature of the changes taking place in the trophic centre? Are they the results of irritation or of destruction of tissue and function? In progressive bulbar paralysis the ganglion cells of the gray matter of certain parts of the medulla, undergo a degenerative atrophy, in the course of which they disappear. There are other changes of minor importance: hyperplasia of the neuroglia, appearance of *corpora amylacea*, and of spider-cells so called, etc.; but the one lesion is the utter destruction of the multipolar ganglion cells. The trophic influence thus withdrawn from the communicating nerve-fibres, they also degenerate, lose their medullary sheath, their axis-cylinder disappears, and nothing finally remains but fibrous cords. In poliomyelitis, we find a similar trophic centre involved, and similar de-

structive lesions. Without occupying time with the intermediate lesions, it suffices to state that the special characteristic of the alterations is the disappearance of the multipolar ganglion cells of the anterior cornua. The connected nerves exhibit corresponding changes, undergo degenerative atrophy, and become little more than connective-tissue cords. The muscles in the case of the destructive lesions of the trophic masses also waste rapidly, their proper anatomical elements disappear, being replaced by fat and connective tissue. The effects in the osseous system are still more extensive and profound. In tabes, typical examples of the joint changes due to lesions of the trophic cells, are described by Charcot in his lectures. As long ago as 1873, Weir Mitchell announced the dependence of bone nutrition on the affection of these trophic cells in this disease, and the occurrence of an abnormal brittleness leading to ready fracture. Peculiar joint affections also occur in connection with cerebral softening, and in certain insane, ribs fracture so readily that, in a trivial struggle with an attendant, one or more may be broken.

The cutaneous trophic lesions, secondary to changes in the gray matter about the posterior nerve-roots, are in a high degree interesting and important, and furnish us with illustrations of the differences between irritation of the trophic centre and its connected nerves, and destruction of function. Furthermore, cutaneous trophic lesions are closely associated with vaso-motor derangements, indicating thus the probable situation of the vaso-motor centre in the spinal cord. The trophic centres for the skin communicate with their end-organs by the sensory nerve-fibres. In destructive lesions of these centres, the proper structure of the skin disappears, and if repair occurs it is only by cicatricial tissue. In the simplest functional disturbance of the trophic centre for the skin, the most transient effects are only discoverable, and the proper structure of the skin remains untouched. There would then seem to be a constant relation between the central and peripheric affections. When there is a mere functional disturbance of the centre of trophic influence, there occurs only a trivial transient skin affection; when there are destructive lesions of the trophic centre, lesions of structure occur at the periphery. Let us see whether these postulates are supported by facts.

Erythema of a few minutes' or hours' duration, urticaria equally fugitive, and some transient cases of herpes, occur from functional irritation of the trophic centre. Herpes zoster, of more or less severe character, is caused by more violent irritation of a sensory nerve

charged with a trophic function. Herpes zoster may also appear in connection with destructive lesions, but in that case there occurs irritation of nerves of temporary duration, and therefore merely an accidental fact, as, for example, the attacks of zoster succeeding to the severe pain of locomotor ataxia. Ecthyma and pemphigus, leading to deep ulcerations of the skin, bullæ penetrating deeply and healing by cicatrix, leucoderma, bronzed skin, etc., are results of destructive lesions of limited extent. Still more significant are the bed-sores, with extensive sloughing, occurring suddenly after a severe spinal or cerebral lesion. In such a case we have exhibited sudden destruction of trophic centres, followed quickly by equally destructive peripheric lesions. No doubt extreme vaso-motor depression coincides with the changes in the trophic centre in such examples.

It is important to ascertain whether in the circuit of trophic action, a disturbance—irritation or destruction of function—at any point will affect the nutrition, or cause lesions elsewhere in the circuit. We know that a disturbance in the trophic centre, and its efferent fibres, will be perceived in changes at the periphery. Does the converse hold true? There are facts, experimental and clinical, which tend to show that destructive lesions of the end-organs cause more or less profound changes in the circuit of trophic influences. Injuries of nerves set up an ascending neuritis, which is only limited by the termination of the connected elements. Friedreich has brought forward a mass of testimony to prove that the profound changes in progressive muscular atrophy are due to an ascending neuritis, beginning in the intra-muscular nerves. Although in this disease the initial change is, by the great majority of observers, located in the multipolar ganglion cells, the facts showing the mode of an ascending neuritis, and the final alterations wrought in the trophic centre, have a high degree of importance in this connection. A clinical fact of immense suggestiveness is the occurrence of ulceration of the duodenum in cases of extensive burn on the chest or abdomen. Very recently I have seen a case of herpes zoster occupying the whole distribution of the right cervical plexus, which was secondary to an attack of hepatic colic, during the course of which there occurred seven distinct paroxysms, due to the passage of as many calculi, proved by their recovery from the feces. The path by which the trophic disturbance was produced was by the solar plexus, the trophic centre in the cervical cord, and the sensory fibres with trophic function of the cervical plexus.

We may now, I think, attempt to apply the principles of trophic action to the pathological states of various organs.

In the cerebral cortex—a collection of psychomotor centres—there is also a centre with a trophic function. There can be little doubt, I think, that such depressing emotions as anxiety, chagrin, and grief exercise an inhibitive influence on the trophic centre, and lead to changes in the cerebral structures and in the mental sphere. To such influences must we refer the early occurrence of senile changes, atheromatous and calcareous degenerations, etc. As in the case of certain joint affections which occur during the course of chronic destructive changes in the trophic centres of the spinal cord, and in which the local inflammation is produced by the urate of soda that collects about the joint, so in certain cerebral states the calcareous salts are abundantly deposited in the brain.

Injuries of certain parts of the brain are followed, as we have seen, by changes in the thoracic organs. Brown-Séquard has made the observation that pneumonia is apt to occur during the existence of disease in the right hemisphere.

The facts showing the influence of the abdominal organs on the brain, and reciprocally, are numerous and important. Of course, I now only have in view the trophic influences. The frequent and persistent occurrence of stomachal vertigo and of migraine is of evil omen, since such subjects, as a rule, suffer early degenerative changes in the brain. The Hippocratic notion, which associated a depressed state of the cerebral functions with hepatic disorder, was not far wrong. Injury of various parts of the brain and cord, of the splanchnics, and of the sciatic may cause glycosuria. Implication of the fibres of the solar plexus, with ultimate destruction of their anatomical elements, is the chief, if not the only, factor in the development of the bronzed-skin disease. An important contribution to the pathology of Bright's disease was made by Prof. Da Costa and Dr. Longstreth when they discovered the profound, degenerative changes in the renal ganglia. Albuminuria is also caused by injuries which involve certain cerebral trophic centres. Injuries involving the splanchnics and the ganglia of the abdominal sympathetic, by paralyzing the vaso-motor fibres, cause swelling of the liver and spleen, and the opposite state is the result of merely irritative action. When the circulatory disturbance is added to the trophic, structural changes quickly follow.

There is a practical question connected with the subject of trophic influences, so pertinent to the subject in hand, that I cannot fail to allude to it here: that is the question of counter-irritation. I have mentioned the fact that, after severe burns of the chest or abdomen,

ulceration of the duodenum occurs in a considerable proportion of the cases; and under the same circumstances, an acute pneumonia or pleuritis may develop. These are pathological experiments of deep significance. We have a right to conclude, from the facts which I have submitted, that the injury in the case of the burn so depresses the functions of the trophic centre that some one of the results mentioned may occur. A large blister, allowed to draw thoroughly, has the effect of a burn. The end-organs of the sensory nerves in the skin are injured, and the trophic centre is depressed. We are aided here in coming to a right conclusion by the results on the urinary secretion of a succession of blisters to the joints in rheumatism. It is, of course, known to all here present that under such circumstances the urine, from being acid, becomes neutral or alkaline. We are further helped to a decision by the observations of Vulpian and others on the effect of injury to afferent nerves on the centre to which they proceed. We cannot localize to the point of injury the mischief done to a nerve, for it passes up through the whole extent to the gray matter. Blisters, large and long in action, are therefore proper only when the functions of the trophic centre are to be lowered. To this might be added, also, when the vaso-motor system is to be depressed. Conversely, transient irritation causes reflex contraction of the vaso-motor fibres, and excites the trophic system to acts that involve function only.

The facts recently discovered by Brown-Séquard in regard to the effect of chloroformic and other kinds of irritation of the skin on the functions of the brain and spinal cord, and the changes in the nutrition of the heart which follow injury to the vagi as demonstrated by Wasilieff, are further evidences of a convincing kind that far-reaching consequences flow from peripheric irritation of the trophic centres.

From every point of view, then, the trophic system has an important place as a pathological factor. I can only regret that I have been unable to add any new facts. Inferences based on facts are, however, not without utility, and these I submit to your reflection.

Dr. Tyson said that no one who had heard the facts adduced by the speaker, and the conclusions arrived at by him, could fail to agree with him that nervous influence had much to do with pathological processes, independent of the existence of special trophic nerves. He said also that he could not forbear calling attention to the fact that almost the entire argument in favor of nervous influence upon pathological processes is based upon clinical pathology, many of the most important experiments, having for their object the demon-

stration of such influences, being open to objection. This may be said of two very notable experiments, already quoted, section of the fifth pair of nerves, and of the pneumogastric nerve, the former of which results in inflammation of the cornea, and the latter in pneumonia. It is well known, however, that if precaution be taken to prevent the access of irritating matters to the eye and lungs, after such section, inflammation does not follow. But no one can deny the influence of the many nervous lesions in the production of results quoted by Prof. Bartholow, such as the effect of spinal lesions on the production of bed-sores, etc.

Dr. Eskridge said that he had had recently a case of abscess of the brain, which seemed to support the views of the lecturer in regard to trophic nerves, and trophic nerve-centres. The abscess was four and a half inches long, and involved the centrum ovale of the right postero-frontal and parietal lobes. The left arm and leg were completely paralyzed, and the muscles of the left side of the face were parietic. The left eye and ear remained normal until just before death, and at no time were there any evidences of inflammation in the external structures of this eye. The right eye and ear, which were on the non-paralyzed side, were the seats of inflammation many weeks before the patient's death. Suppuration destroyed the function of the right ear, and inflammatory thickening of the cornea and conjunctiva vision of the right eye. At the post-mortem examination the base of the brain and optic nerves were found to be in an apparently healthy condition. There were swelling, redness, and great induration of the right side of the neck, which must have interfered with the sympathetic nerve and ganglia in this region.

He would like to hear the views of Prof. Bartholow a little more in detail on the subject of counter-irritation. He had understood him in the course of his lecture to state that mild counter-irritation increased, and severe (such as blistering) decreased the function of the deeper structures beneath the counter-irritant. If this theory is correct, mild counter-irritation (such as mustard-plasters, etc.) was injurious in the first stage of inflammation, and blistering was contra-indicated during the stage of effusion. He concluded from the doctor's views that blisters, if used at all, should be applied early in acute inflammations, and that mild counter-irritants were indicated during the stage of effusion, or when the resolving process of inflammation was taking place slowly and imperfectly.

Dr. Formad said he had profited a great deal from Prof. Bartho-

low's interesting paper, and was glad to hear that the lecturer, if he understood him aright, did not consider nerve action an important factor in the etiology of inflammation. Although this question was not directly pertaining to the scope of the paper under discussion, Dr. Formad thought it interesting to bring forward in this connection a few well-known facts concerning the neuro-pathology of inflammation, and also to point out some sources of errors in the pathological anatomy of the nervous system.

In text-books on surgery various inflammatory processes are mentioned as being produced by nerve influence; and in clinical medicine, also, too much stress is laid upon the direct action of the so-called trophic nerves. It is certainly incorrect to connect the action of nerves with the process of inflammation, except in a merely indirect manner. All the beautiful experiments, made lately by various investigators, appear thoroughly to overthrow the phlogogenic influence of nerves. Dr. Formad desired to enumerate only a few of the experiments, those which he had had opportunity to corroborate himself. In the pathological laboratory of the University of Pennsylvania, Dr. Francis Dercum and he had repeated many times the experiments of Schnellen and Senffleben, of section of the trigemini and sympathetic nerves, and the classical experiments of Traube with the pneumogastric nerves. As routine work of the laboratory course, these and other experiments in neuro-pathology were also made every year by his students, and, under proper care, give always identical results. Section of the cervical portions of both pneumogastric nerves always promptly gave rise to pneumonia in the animal experimented upon. But it could distinctly be traced that the croupous pneumonia and trachitis were purely traumatic, produced by irritation from particles of food, etc., which had entered the air-passages. What the nerve section had effected was merely a paralysis of the epiglottis and vocal cords, which, not being capable of closing, allowed the entrance of the foreign particles mentioned. The same is proven for the keratitis, following section of the trigeminus. The main effect on the eye of section of the fifth nerve is paralysis of the corresponding eyelids, and probably the checking of secretion of the lachrymal gland. The eye is constantly open and dry; it is also insensible and fixed. Still, if we protected such an eye by a suitable covering, we succeeded a number of times in preserving it for a long while free from any signs of inflammation. If, however, the cover was removed, or not applied at all, inflammation set in at once. The direct cause of the inflammation

is here evidently to be looked for in the impurities and the drying effect of the air, and in direct injuries, the animal knocking its insensible, unprotected eye against obstacles. Again, without the severance of any of the nerves, a violent keratitis is soon induced, if the eye is kept constantly open by means of a suitable apparatus to prevent the closing and the motion of the eyelids.

Various other inflammatory lesions are brought into the domain of neuro-pathology, but none of them stand the test, the only scientific test, that of direct careful experiment.

Neuro-pathology has a greater and more justifiable application in lesions depending upon retrograde changes, such as the atrophies and the degenerative processes of various kinds. Dr. Formad thought it probable, however, that even here nerve influences, as direct factors, were largely overrated, and that objection can be raised to many of the discoveries in neuro-pathology. For many lesions in the nervous system, the proper interpretation of the cause and of the anatomical changes is not made. Nerve-tissues, like the brain and spinal cord, are subject to most remarkable post-mortem changes through accidental influences, and there is no field in pathological histology so apt to mislead the observer, and no more fruitful field for hallucinations than the histology of the nervous system. Dr. Formad said that he had heard Virchow himself say that one-half of all the discoveries of lesions of the nervous system were mostly due to post-mortem changes, and were artificial. Dr. Formad had had ample opportunity to convince himself of this truth, and admitted himself having made mistakes of this kind. Experiments made in his laboratory have shown a decided morphological difference between a brain or cord put into proper preserving fluid immediately after death; and similar specimens that were obtained from an autopsy executed twenty-four to forty-eight hours after death, or in specimens which were kept in unsuitable preserving fluids. The temperature in which the bodies were kept also remarkably influences post-mortem changes in the brain and cord. More precautions have to be taken with these than with any other tissues. All kinds of atrophic and degenerative changes are produced artificially by mere neglect or ignorance of the methods of hardening and mounting of such specimens; and it is here where some of the discoveries in nervous pathology, such as many of the softenings, atrophies, amyloid changes, etc., belong. The mistakes and hallucinations of the incompetent microscopist and experimenter mislead the clinician, and have largely

contributed to erect the shaky building of Neuro-Pathology and of the trophic action of nerves.

Dr. Formad did not mean, however, to raise any question at all concerning the observations of experienced men, but such, he thought, were few in the domain of the pathological anatomy of the nervous system.

April 28th, 1882.

7. *Case of erysipelas neonatorum.*

Presented by Dr. H. M. FISHER.

I was called, March 3, to see a colored infant, one week old. The child was said to have presented no sign of indisposition until the day previous to my visit, when it suddenly became much agitated, refused to move, and was found to have difficulty in micturition. The tissues of the scrotum and penis were found to be swollen, and in a few hours a faint erysipelatous blush was found pervading their surfaces.

When first seen, the child was apparently moribund. I could detect no radial pulse, its lips were blue, its extremities cold, and it was in a semi-comatose condition. In the hasty examination I made in the small, badly-lighted attic, in which I found the child, I observed, but did not attach due importance to, the erysipelatous blush I found extending from the scrotum to the lower part of the abdomen. From the history furnished me, and from the œdematous infiltration of the legs, thighs, and eyelids, as well as of the scrotum and penis, I was inclined to look upon the case as one of primary acute nephritis, with secondary erysipelatous involvement of the œdematous tissues.

With this view of the case, I ordered that the child should at once be placed in a hot bath, and after the bath applied flannels, wrung out in hot infusion of digitalis, to the patient's loins, and administered a small hypodermic of pilocarpine. Marked temporary improvement followed this treatment; the child emerged for a short time from its semi-comatose condition, cried lustily, and urinated and sweated freely, and marked reduction of the œdema was also noticed.

The improvement was, however, of short duration; the child soon relapsed into its former apathetic condition. Death occurred at 11 P. M. The autopsy, made by Dr. McIlvaine, resident surgeon, eighteen hours after death, revealed rigor mortis pretty well established.

Edema of the tissues of the lower extremities, scrotum, and penis was noticed; there was a livid discoloration of the skin of the anterior

inner aspects of both thighs, and slight sloughing of the skin of the lower part of the scrotum. The bladder contained about half an ounce of light-yellow opaque urine. The urine obtained by tapping the bladder, post-mortem, showed, on examination, an apparent volume of albumen, equal to about one-eighth of the column of urine in the test-tube. Microscopically, blood- and pus-corpuscles were found in abundance, and a few highly granular tube casts. The kidneys were somewhat congested and lobulated, but their capsule was not adherent, and they did not, at least to the naked eye, present any other evidence of disease. The liver weighed six ounces, and was highly congested. The spleen weighed one and a half ounce, and was likewise congested, and its surface mottled. The lungs were pretty evenly congested throughout, but were everywhere crepitant, and floated on water.

The heart weighed two ounces, and presented, on the whole, a healthy appearance, but its posterior mitral cusp appeared somewhat thickened.

The post-mortem appearances leave, I think, but little doubt that the disease was peripheral in its origin, or, in other words, that the erysipelas was the starting-point of it. Probably some comparatively trifling cause, such as a slight abrasion of the skin of the scrotum, sufficed, under the very unfavorable sanitary conditions in which the little patient was placed, to light the train of its disease, with all its complications.

The progress of the disease seems in this particular case to have been unusually rapid. If the mother's statements can be trusted, only thirty-six hours elapsed from its first onset till its fatal termination.

March 22d, 1883.

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